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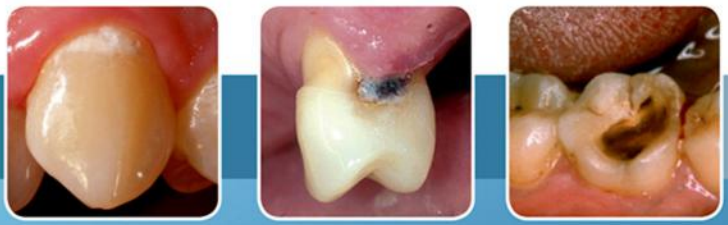
Essentials of Dental Caries



Fourth edition

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Essentials of Dental Caries:

Essentials of Dental Caries:

FOURTH EDITION

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Prologue

Welcome to the 4th edition of *Essentials of Dental Caries.* Sally Joyston-Bechal, one of the original authors, retired some years ago and Edwina Kidd has enlisted the assistance of an old friend and colleague, Ole Fejerskov, to put fingers to keys to write this last effort with her. The collaboration is appropriate as we are editors of a rather large and heavy, multi-authored text on Cariology, now in its third edition and selling worldwide. The original aim of *Essentials* was to write a small, straightforward book suitable for junior students, dental nurses, oral health educators, hygienists and therapists, and other oral health care workers. We are proud of our large textbook, but feel it may be a daunting place to start the study of this very important subject. Hence, an attempt to write a much shorter text, with the hope that the dental student, in addition to the other oral health personnel, may read this while at the beginning of their studies, but progress to the multi-authored, more comprehensive book before qualification and at postgraduate level.

We have given very few references as our aim is to provide the reader with a coherent text focusing on what we think is **essential**. We have distilled current evidence-based knowledge for the reader and, in 'Further reading' at the end of each chapter, point to fully referenced articles and chapters.

We come from two European countries (United Kingdom and Denmark), but we have tried to make this text applicable in other English-speaking countries worldwide. This is not easy as, to state the obvious; the different economic conditions over the world make it a problem to write recommendations that are affordable and practical in all countries. Moreover, in the process of writing we have realized that even between our two northern European countries, the marked differences in the way in which the different countries have historically organized their oral health care, seems to play a surprising role in the way the dental professionals are trained and influenced during their years of study. Therefore, throughout the text we have tried to make new readers aware of the fact that there does not exist only one way that represents the 'truth'. We have interpreted the scientific data and written what, hopefully, is a coherent text, constantly asking ourselves, 'What are the consequences of these data for oral health care in the population? How can oral health for every individual best be further improved, as cost-effectively and simply as possible?'

So how is this small text organized? In Chapter 1 the extreme importance of the subject to dentistry is illustrated. The consequences of dental caries occupy most dentists and ancillary personnel for most of their time. Chapter 2 describes how the caries lesion develops and progresses. It introduces the microbial biofilm and explains how bacteria, which are essential partners of human beings, are able to metabolize sugar in the oral cavity, produce acid as a waste product, and possibly demineralize teeth so that caries lesions are obvious. Fortunately, most of us have sufficient saliva, a 'healing fluid', containing the necessary minerals to control this damage. The central role of sugar is emphasized and why it so far has been so difficult to restrict sugar intake is discussed.

The appearances of lesions are described and illustrated in Chapter 3 with emphasis on the diagnosis of active lesions. These signify the proven risk of developing further lesions. Chapter 4 considers the control of lesion development, for everybody, and concentrates on the roles of oral hygiene, fluoride, and diet. It is important to fully understand this chapter in order to manage the problem sensibly. The chapter also considers who is influencing the global sugar market place.

Chapter 5 introduces the concept of filling teeth, showing when this is required to aid cleaning and thus arrest the lesion. It is stressed that fillings are not a treatment for the disease dental caries, but part of facilitating the patient's cleaning. It is this cleaning that is the key to caries control. When patients develop caries lesions they need to change both cleaning habits and diet. Behaviour change is salient to caries control and so Chapter 6 discusses the difficulties of behaviour change, describing one method in some detail. These techniques rely on effective communication with the patient and how this might be done is discussed.

Chapter 7 comes right to the heart of the caries control problem by describing how to explain why an individual patient presents with caries lesions. What is special about this mouth, what needs to change? The text then concentrates on patients who present with active lesions, and looks at oral hygiene, diet analysis, and advice, and how to most sensibly apply fluoride in this caries active group. Finally, it addresses the problems of specific groups, including the very young and the very old.

Thus far, the book has concentrated on the individual patient, but Chapter 8 changes focus to look at communities and introduces how data on caries is collected in communities. Finally, it is shown how one community in Denmark used this information to change the way patients were cared for, then analysed the results of these changes. Moreover, the approach taken in Scotland is examined. This exemplifies that there are different ways to cope. In our view one should always aim for the most simple, cheap, and effective way to serve the need of populations.

We conclude the book with a very personal Epilogue. Here we turn conventional dental wisdom on its head and encourage the reader to think differently about the dental personnel required to deliver caries control cost effectively. We do not think this is best done by dentists as they are currently trained, and we dare to suggest that, with today's knowledge of the reasons for and progression of the major oral diseases, we may be training far too many dentists! Enjoy and discuss, and please feel free to contact us with questions and for an exchange of knowledge.

Edwina Kidd and Ole Fejerskov
London and Aarhus in February 2016

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Chapter 1

Introduction

1.1 The goal of oral health care

A pain-free, functioning, and good-looking dentition for a lifetime seems a reasonable goal! Is this what dentists do? An advertisement for a North American dental practice recently suggested that dentists practising general dentistry provide amalgam and composite fillings, sealants, cosmetic dentistry, pulp and root canal treatment, crown and bridges, dentures, and dental implants. Moreover, they do minor oral surgery, gum disease treatment, and occasionally temporomandibular joint (TMJ) therapy, tobacco cessation, and nutrition counselling. The topics listed in the first sentence comprise the daily work in general dentistry, **but do you realize that 85% of these are a direct consequence of dental caries?** Yet dental caries is not mentioned as the main reason for most dental treatments. Restorative treatment is the focus of dentistry. The disease dental caries is the only disease which has been combatted with metals and composites for more than a century.

Some 50 years ago the concept of prevention became fashionable. Now restorative treatment was described as 'secondary prophylaxis' because it was considered that once the inevitable dental caries had occurred, it had to be treated (i.e. restored) to prevent further break down of the teeth and the dentition. Therefore, it is not surprising that the most time in the dental curriculum is devoted to the many skilled restorative procedures. These have to be conducted in a moist, slippery, small, and moving oral cavity attached to a person who may find the procedure unpleasant! No wonder it is difficult to perform intra-oral restorative work of high quality as part of oral rehabilitation, and no wonder so much time in the curriculum is devoted to these aspects.

However, supposing it was possible to prevent or control the disease so that restorations are reduced to a minimum? This control of caries is what this book is about! Seven chapters present the essentials of what is known about dental

caries. The observations will be based on current scientific evidence. This is a hands-on book, which means that what is suggested and observed should have immediate implications for how patients may be treated. By 'treatment' we mean all procedures that control the disease, including restorative work, but restorative work is not the focus of the effort. Above all, there is no separation of prevention and disease control from 'treatment' (i.e. restorations). Case stories from daily practice are presented, but the proposals and recommendations will be based on the outcome of controlled clinical trials, carefully conducted clinical studies and population studies. In other words, the recommendations are based on data that document a 'significant' (i.e. large) effect in populations. At the outset, however, the relative importance of dental caries in oral health must be considered.

1.2 Why do patients lose teeth?

This is a logical question if the goal of oral health care is to preserve the teeth. About half a century ago it was common to claim that children were particularly 'caries active'. Once they became an adult they experienced much less dental caries, but when they aged further, with partly exposed root surfaces, another caries active period occurred. This is incorrect, but to understand how such misconceptions developed the oral health condition prevailing at the middle of the previous century must be appreciated. Tooth cleaning was predominantly performed to achieve a 'white smile' and remove stains from the teeth. Unfortunately, large amounts of microbial soft and hard deposits were present in children who were fed on a sugar-rich diet with frequent snacks between meals. **With the knowledge currently available**, it was to be expected that children would experience a high rate of caries lesion development. In northern Europe it was common that, by the age of 12–14 years, children would have had several teeth extracted. In poorer societies along the west-coast of Denmark it was even common that 14-year-old girls, as a gift for their confirmation, had all teeth extracted and replaced with full dentures. Problems solved once and for all, it was thought! There were rather few dentists outside the major cities and they could not cope with the caries situation. They spent most of their time drilling and filling cavities with amalgams. Most adolescents had one or more teeth extracted, and most remaining surfaces (occlusal and approximal) filled, so no wonder that 'new' sites at caries risk were few once they reached about 18 years old.

Of course, new caries lesions developed along the margins of fillings because these were plaque covered. Unfortunately, dentists interpreted this as their fault, the margins, or the dental materials, were not good enough and favoured 'secondary or recurrent' caries development. For many years secondary/recurrent caries was thought to be something different from 'primary' caries.

As well as these restorative efforts to control disease, public school dental health programmes were developed in many countries. Here, the focus was on prevention, as well as restoration, and the importance of fluoride in the prevention of caries became popular, based on the interesting studies coming from the USA in the 40s and 50s. At this stage, it was thought that tooth loss before the age of 30 years was a result of caries.

However, after the age of 30 years it was claimed that periodontal diseases were the main cause of tooth loss! This concept is incorrect, but to appreciate how it arose, again, the understanding of the natural history of the two major oral infectious diseases (caries and periodontal diseases) a few decades ago must be considered. Most restorative dentists (and so-called cariologists) focused on improving dental materials and techniques to avoid secondary caries. The cariologists focused on diet, fluorides, and mutans streptococci, the organisms considered to be mainly responsible for caries. At this time, the rapidly growing discipline of periodontology totally 'monopolized' the roles of dental plaque and oral hygiene in the oral environment. During the last five decades, the relative role of dental plaque for the development of caries has been debated and the importance of oral hygiene in caries prevention is, even now, frequently questioned, and this will be returned to in detail in subsequent chapters.

Around 1980, observations from large population surveys in low income countries in Africa, South-east Asia, and China challenged these dogmas. These populations had very poor oral hygiene, and often no or very limited access to dentists and dental health care. The oral conditions in adults and the elderly looked unpleasant, with extensive accumulations of soft and hard deposits on teeth and along the gingival crevices (Figures 1.1 and 1.2). Gingivae were inflamed and swollen, often bleeding, and with age there was a gradual gingival recession. Caries lesions were present, at different stages of breakdown,

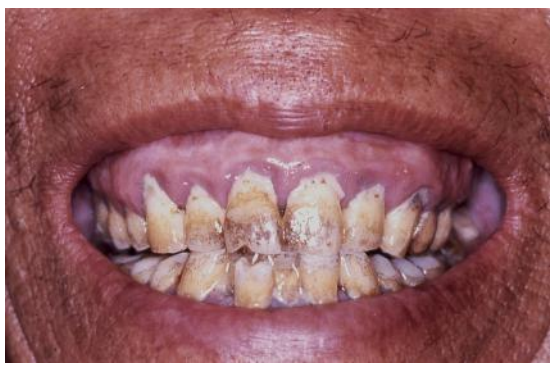

Figure 1.1 Oral condition in an elderly Chinese man.

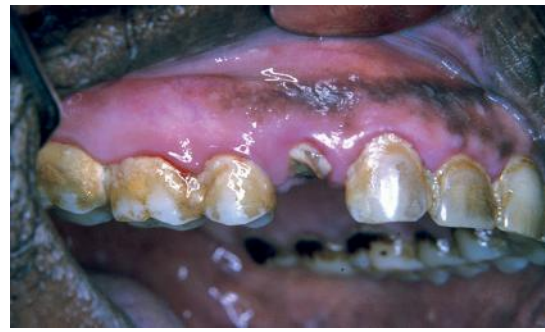

Figure 1.2 Massive dental plaque in an adult Kenyan man. The oral hygiene among these populations is very bad with swollen gums, but people rarely lose their teeth because of periodontal loss of attachment. Dental caries is the principal cause of tooth loss.

but there was a limited loss of teeth. The teeth lost were lost predominantly because of deep caries, because caries progression and initiation continue for the whole of life. Periodontally, there is a gradual loss of periodontal attachment accompanied by gingival recession, but the number of teeth extracted because of periodontal disease is **much** lower than those extracted because of untreated caries.

So the important conclusion is that, if not dealt with, caries lesion development and progression continues for the whole of life, and is the main reason for loss of teeth in populations with no or limited access to dental health care.

In the 1980s, in high-income countries with a large number of dentists, the patterns of tooth loss were much more extensive than that described above. For example, in Denmark, where there are about 1200 patients per dentist, almost 60% of the adults above the age of 60 years had partial or full dentures, and the amount of restorative treatment was overwhelming. In Figures 1.3–1.6 examples of the oral conditions in adults are presented.

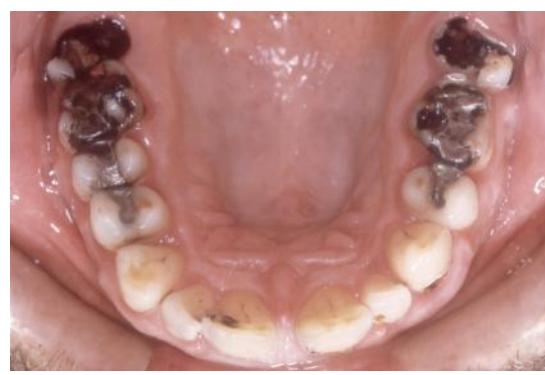

Figure 1.3 Fifty years ago Danish and English adults, including those with deep caries lesions in molar teeth, had their teeth repaired using dental amalgam. The figure shows poor oral hygiene, old amalgam restorations, fractured teeth, and new lesions formed adjacent to the fillings. Re-restoration is needed.

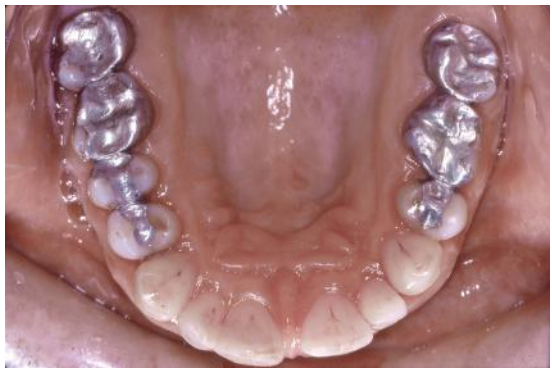

Figure 1.4 The restorations in Figure 1.3 have been replaced, but even these very nice restorations will deteriorate over time if there is no proper oral hygiene as part of caries control. Hence, the vicious circle of re-restoration continues.

Chapter 5 explains why restorative dentistry has become such a burden to societies and has resulted in the so-called restorative cycle, which inevitably leads to loss of teeth.

So it must also be concluded that dental caries and restorative care are by far the predominant causes of tooth loss in high income countries.

It is now apparent that dental caries and its sequelae is the main reason for loosing teeth in all populations. Dental caries is central to the oral health need of populations, and it is ubiquitous in all populations. Next, what dental caries actually is will be discussed.

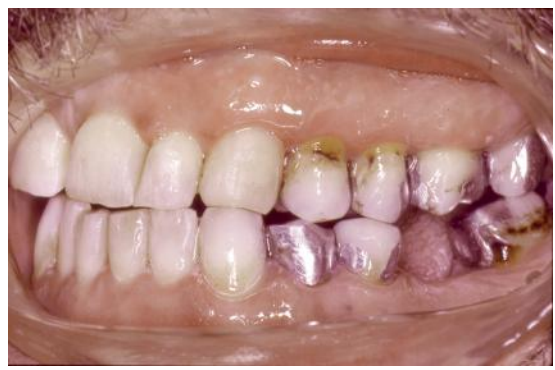

Figure 1.5 Adult patient with an extracted lower first molar and amalgam restorations. The remaining amalgams have been polished and the patient has been carefully instructed on how to control dental caries. Therefore, there are several arrested, almost black, caries lesions in the exposed root surfaces of the upper premolars and lower second molar.

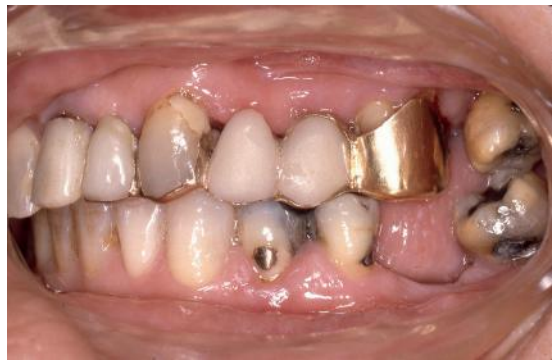

Figure 1.6 An adult who has so far not had appropriate caries control and has entered the restorative cycle. In the upper jaw, the central incisor has an ill-fitting jacket crown. There is a 5-unit bridge replacing the premolars and a plastic restoration is present on the distal surface of the canine. Here, the dentist has probably tried to repair a secondary caries attack next to the gold. The second molar has been extracted. In the lower jaw the first molar has also been extracted. Buccal amalgam restorations are present on the second premolar and molar, but notice the one in the molar is plaque covered. There is a gold foil restoration on the first premolar. These are time-consuming to insert and therefore expensive, which indicates that this patient has actually invested in restorative care. What will happen to this dentition over the next 5 years?

1.3 Dental caries—a definition

It is a localized, chemical dissolution of a tooth surface brought about by metabolic activity in a **microbial deposit** (a dental biofilm) covering a tooth surface at any given time. The dental biofilm (called dental plaque) is a microbial biomass composed of resident bacteria from saliva (see Chapter 2). The dental biofilm is disturbed when brushing the teeth. The micro-organisms metabolize sugars from the diet and, as a waste product, produce acid. This acid can demineralize enamel, dentine, and cementum, and the lesions manifest themselves clinically in a variety of ways as will be described.

Dental caries lesions may develop at any tooth site in the oral cavity. There are no parts of a tooth that are 'more resistant' or 'less susceptible' to developing caries lesions due to variations in the chemical and structural composition. Dental caries lesions develop at relatively 'protected sites' in the dentition, where dental biofilms are allowed to accumulate and mature over time. Such sites include pits, grooves, and fissures in occlusal surfaces, especially during eruption, approximal surfaces cervical to the contact point/area, and along the gingival margin. Obviously, insertion of foreign bodies into the dentition

(e.g. fillings with inappropriate margins, dentures, orthodontic brackets) may also result in such 'protected' sites. These areas are relatively protected from mechanical influence from the tongue, cheeks, and abrasive foods, and not least, toothbrushing. Thus, these are the sites where lesion development is more likely to occur because the biofilm is allowed to stagnate there for prolonged periods of time. Dental caries lesions, furthermore, do not develop at the same rate in all parts of the mouth. Thus, openings of the major salivary glands represent areas with a special salivary composition, which favours a relative protection towards chemical dissolution because of buffering capacity and chemical composition of the secretory product.

Dental caries lesions result from a shift in metabolic activity accompanied by a gradual change in ecology of the dental biofilm, where an imbalance in the equilibrium between tooth mineral and biofilm fluid develops. It is important to appreciate that an oral biofilm, which forms and grows ubiquitously on solid surfaces in the oral cavity, does not necessarily result in the development of clinically visible caries lesions when grown on a tooth surface. However, the biofilm is a prerequisite for caries lesions to occur. Dissolution (demineralization) occurs when acid forms and biofilm pH drops below a certain level. Redeposition (remineralization) of minerals occurs when biofilm pH goes up again. These changes take place at the interface between the biofilm and the tooth surface numerous times during a day, and can be modified extensively.

Over time, the outcome of these fluctuations may result in a **disturbance of the equilibrium** between the tooth mineral and the surroundings. Mineral loss, subsequent lesion formation, and possible cavity formation in the teeth, is a **symptom of the imbalance** in this dynamic process. The metabolism in the biofilm is an ubiquitous, natural process—part of having teeth. However, its possible consequence—lesion formation and progression—**can be controlled** so that a clinically visible lesion never forms or an established lesion is arrested.

If, for example, the biofilm is partly or rarely totally removed by toothbrushing, mineral loss may be arrested or even reversed towards mineral gain because saliva is supersaturated with respect to the enamel apatite. This will result in the arrest of disease progression and may even result in some redeposition of minerals in the very surface of the tooth. If, on the other hand, diet changes with, for example, higher sugar intake (numerous sweet drinks, sweets, cakes, etc.), the ecology of the biofilm changes. Acid-producing bacteria multiply and demineralization predominates.

Any factor that influences the metabolic processes, such as the composition and thickness of the biofilm, the salivary secretion rate and composition, diet, and fluoride ion concentration in the oral fluids, will contribute to the balance

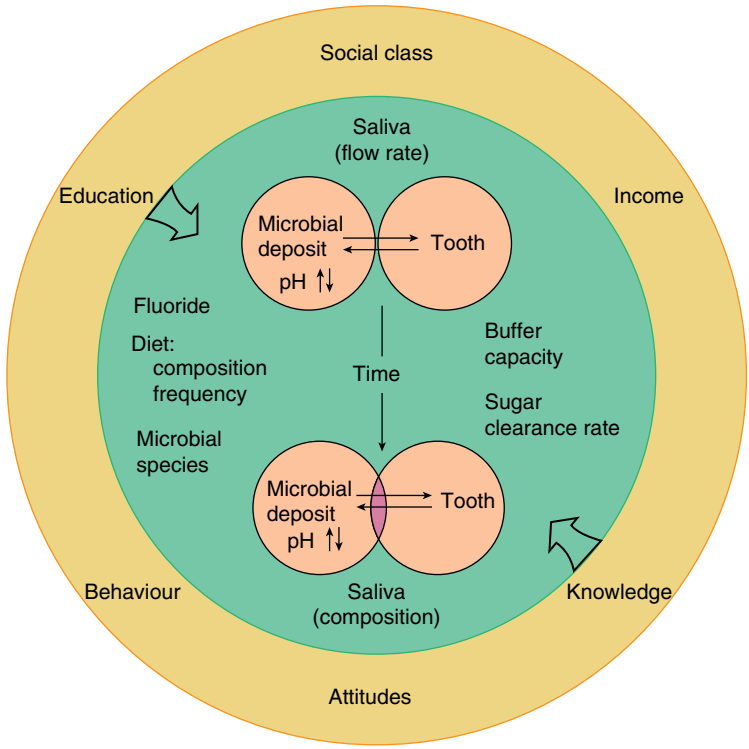

Figure 1.7 Some of the most important variables that determine the relative rate by which caries lesions may develop. The teeth are covered by microbial deposits (the inner small circles interacting over time). The microbial deposits are the same as dental plaque seen in Figures 1.1 and 1.2. Today dental plaque is also called 'dental biofilm'. The metabolism in these microbial deposits is reflected by pH fluctuations; over time this may result in a caries demineralization in the tooth (the hatched area between the lower circles). If and when this happens is highly influenced by a variety of factors in the oral cavity, the most important being saliva, fluoride, diet, microbial species, buffer capacity, and sugar availability. The inner big circle reflects the oral cavity, while the outer circle shows a variety of social factors in populations that indirectly influence the likelihood of the development of caries lesions.

between net loss or gain, of mineral – and the **rate** at which this occurs. Figure 1.7 indicates how the many biological determinants of the caries process may act at the level of the individual tooth surface (inner circle). At the individual/population level (outer circle), the behaviour, education, knowledge, and attitudes will have a strong influence on some of the biological determinants (quality of oral hygiene, choice of foods, use of fluorides, salivary flow from chewing gum, etc.). All these parameters will be discussed in the subsequent chapters.

At any given point in time, the net mineral loss or gain is part of a continuous spectrum of events. The absence of a clinically detectable caries lesion does not necessarily mean that no mineral loss has occurred—it only means that it could not be discerned clinically. If this concept of a continuum is appreciated it will immediately be understood why detection of various stages of lesion progression is a question of defining certain 'cut-off' points along the continuum.

1.4 Classifying lesions

A number of ways of classifying lesions will be used in this book. For instance carious lesions are classified according to their **anatomical site**. Lesions may commonly be found in **pits and fissures** (Figures 1.8 and 1.9) or on **smooth surfaces**. Smooth surface lesions may start on enamel (**enamel caries**; Figures 1.10 and 1.11), or on the exposed root cementum and dentine (**root caries** Figures 1.12 and 1.13).

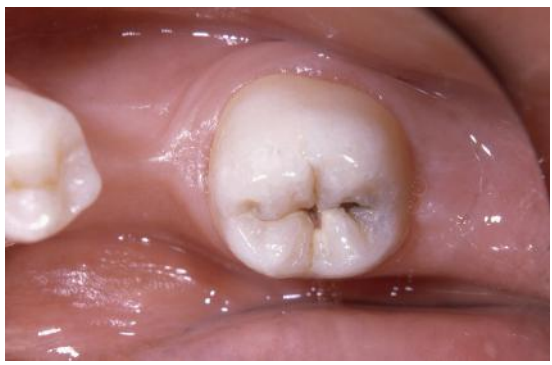

Figure 1.8 Active fissure caries on the occlusal surface of a molar. The lesions are cavitated.

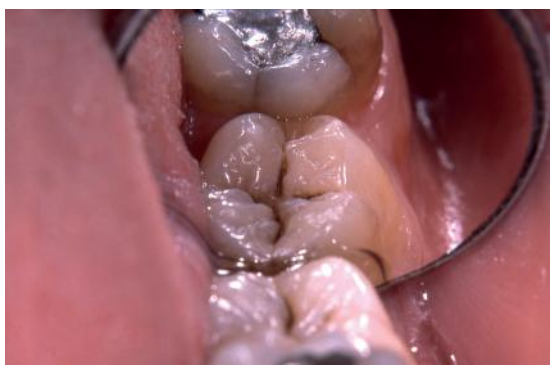

Figure 1.9 Inactive occlusal surface caries. These lesions are not cavitated.

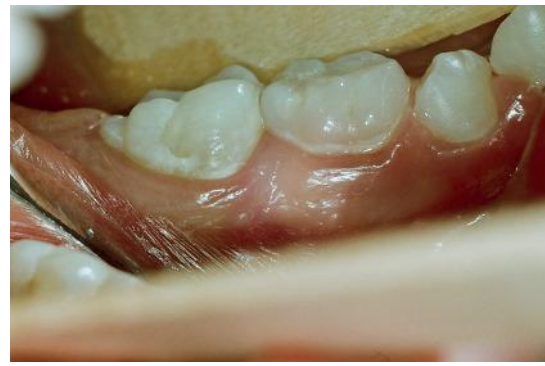

Figure 1.10 Buccal surface with active enamel caries lesion. The early stages often appear chalky white (also called 'white spot' lesion).

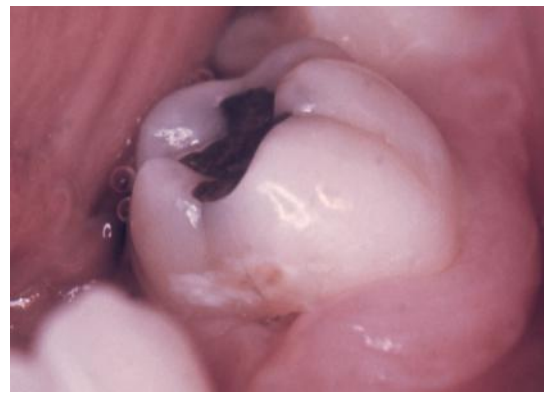

Figure 1.11 Inactive or arrested caries lesion on an approximal surface. Such lesions are often partly stained.

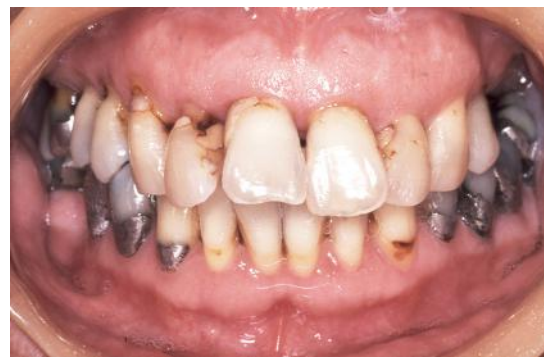

Figure 1.12 Heavily restored dentition with many inappropriate restorations, where new caries lesions continue to develop as the root surfaces are exposed. These new lesions at the margins of the restorations are called secondary or recurrent caries, and they are then restored.

Primary caries is used to differentiate lesions on natural, intact tooth surfaces from those that develop adjacent to a filling, which are commonly referred to as **recurrent** or **secondary caries** (Figures 1.12 and 1.13). As already implied, the only difference between recurrent or secondary caries and primary caries is whether a filling is present lying adjacent to a lesion. Recurrent caries should be

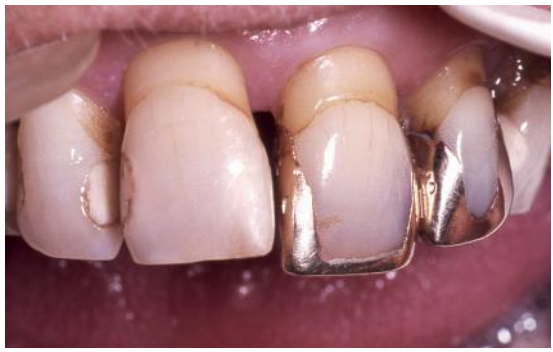

Figure 1.13 The patient has invested in expensive gold restorations. However, a new lesion is developing on mesial surface of the lateral incisor. Note the staining (the patient is a smoker!) of the margins of old silicate fillings and along the enamel-cementum junctions.

differentiated from **residual caries**, which as the term implies is demineralized dentine that has been left behind before a filling is placed.

A lesion is either **cavitated** or **non-cavitated.** A cavity is a physical hole in the tooth and may impinge directly on the management of the lesion.

Caries lesions should, in the authors' view, be classified according to their activity, irrespective of being cavitated or not. This is a very important concept and one that impinges directly on management, although it will be evident from the text that the clinical distinction between **active** and **inactive** (arrested) lesions is sometimes difficult.

A lesion considered to be progressing (it is anticipated that the lesion would have developed further at a subsequent examination if not interfered with) would be described as an **active caries lesion**. This distinction is based on a judgement of the features of the lesion in question, in combination with an assessment of the oral health status of the patient. In contrast to this is a lesion that may have formed years previously and then has not progressed further. Such lesions are referred to as **arrested caries lesions** or **inactive caries lesions**.

You may also meet the terms **remineralized** or **chronic lesions** used to signify arrested lesions; but, as will be appreciated later, the term remineralization should be used with caution. The distinction between active and inactive/arrested lesions may not be totally straightforward. Thus, there will be a continuum of transient changes from active to inactive/arrested and vice versa. A lesion (or occasionally part of a lesion!) may be rapidly progressing, slowly progressing, or not progressing at all. This will be entirely dependent on the ecological balance in the biofilm covering the site and the environmental challenge. Clinically, if in doubt, the dentist should always react as if he/she is dealing with an active lesion.

Despite the diagnostic difficulties these distinctions are very important to the clinician because if a lesion is not active, no action is needed to control further progression. If, on the other hand, a lesion is considered active, steps should be

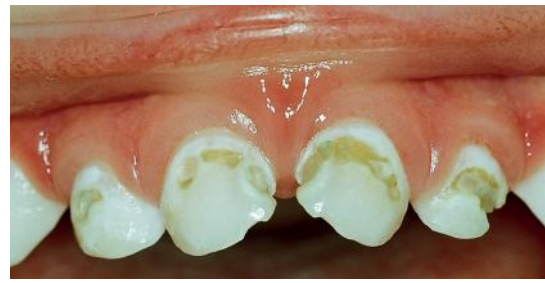

Figure 1.14 Rampant caries in a child. These are deciduous teeth.

taken to influence the metabolic activities and possibly the ecological balance in the biofilm in favour of arrest, rather than further demineralization.

At this point it is also sensible to discuss a possible confusion in terminology. The first sign of a carious lesion on enamel that can be detected with the naked eye is often called a **white spot lesion** (Figure 1.10). This appearance has also been described as an early, **initial,** or **incipient lesion**. These terms are meant to say something about the stage of lesion development. However, a white spot lesion may have been present for many years in an arrested state and to describe such a lesion as early would be inaccurate. A dictionary definition of 'incipient' is 'beginning; an initial stage'. In other words, an initial lesion appears as a white, opaque change (a white spot), but any white spot lesion is not incipient!

Rampant caries is the name given to multiple active lesions occurring in the same patient (Figures 1.14 to 1.15). This frequently involves surfaces of teeth that do not usually experience dental caries. Sometimes the patients with rampant caries are classified according to the assumed causality, e.g. bottle or nursing caries in children, and bakers' caries, radiation caries, and drug-induced caries when seen in adults. Early childhood caries (ECC) is caries in the age group 12–30 months, and there is a specific pattern that differs from caries in

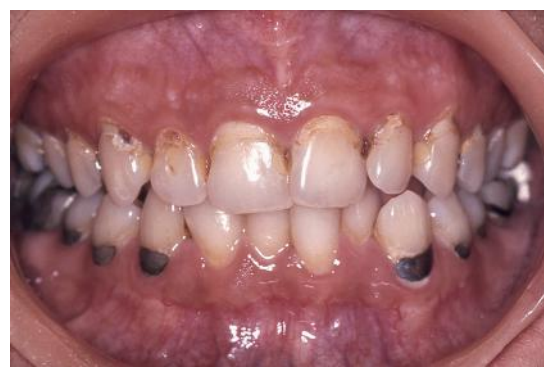

Figure 1.15 Rampant caries in an adult. The patient was a baker and such lesions have been designated 'baker's caries'.

older children. The upper incisors are the most vulnerable (Figure 1.14). This is simply caries on teeth that are not clean, exposed to carbohydrates, and located in an area of the mouth, where oral clearance is low; for an explanation of this see Chapter 2.

Hidden caries is a term used to describe lesions in dentine that are missed on a visual examination, but are large enough and demineralized enough to be detected radiographically. It should be noted that whether a lesion is actually hidden from vision depends on how carefully the area has been cleaned and dried, and whether an appropriate clinical examination has been performed.

In order to be able to control dental caries it is necessary to understand how a lesion develops and progresses with time if not interfered with. This will be discussed in Chapter 2.

Further reading

Baelum, V., Luan, W-M., Chen, X., and Fejerskov, O. (1997) Predictors of tooth loss over 10-years in adult and elderly Chinese. *Community Dent. Oral Epidemiol.*, **25**, 204–210.

Fejerskov, O. and Kidd, E. (eds) (2008) *Dental Caries. The Disease and its Clinical Management* (2nd edition). Blackwell/Munksgaard, Oxford.

Fejerskov, O. and Manji, F. (1990) Risk assessment in dental caries. In: Bader, J. (ed.) *Risk Assessment in Dentistry* (pp. 215–217). University of North Carolina Dental Ecology, Chapel Hill, NC.

Manji, F., Fejerskov, O., Baelum, V., Luan, W-M., and Chen, X. (1991). The epidemiological features of dental caries in African and Chinese populations: implications for risk assessment. In:Johnson, N. W. (ed.) *Risk Markers for Oral Diseases*, Vol. 1 (pp. 62–100). Cambridge University Press, Cambridge.

Chapter 2

How does a caries lesion develop?

2.1 The dental biofilm

The oral cavity is an open sink. The mucous membranes and teeth are constantly covered with a salivary film whose proteins adhere to all surfaces in the mouth. Saliva is not just a fluid flushing through the oral cavity, but a highly complex proteinaceous liquid that contains millions of microorganisms (bacteria). Depending on their different surface properties (different species have different surface proteins comprising their cell wall, which coat the surface of each cell) they stick to the salivary proteins at the surfaces of mucous membranes and teeth. These oral microorganisms comprise the **endogenous** flora of the mouth. They are living in symbiosis with the cells of the human body and comprise what is today called the metagenome.

There are more bacteria covering all body surfaces in each individual than there are eukaryotic cells in the whole body. Eukaryotes store their DNA in a membrane 'sac' called the nucleus. Plants, fungi, and animals are eukaryotes, whereas bacteria are prokaryotes with no distinct nuclear compartment in which to store their DNA.

Prokaryotes live in a variety of ecological niches. An occlusal fissure is an example of such a niche and so is an approximal space between neighbouring teeth, the gingival crevice, and periodontal pockets. Bacteria are astonishingly

varied in their biochemical capabilities—in fact, more so than eukaryotic cells and each ecological niche may have a particular environment (different pH, inflammatory exudate, etc.), which will influence the microbial function and composition.

Until recently, traditional bacteriological methods were used to isolate and culture microorganisms in the laboratory, but it was realized that only a few could be cultivated! DNA sequencing techniques (genomics) of populations of microorganisms from a variety of natural habitats (including the oral cavity) showed that most species have not been found by these traditional culturing techniques. **According to some estimates, about 99% of prokaryotic species remain to be characterized**.

For this reason alone, it does not make sense to think that a particular 'caries microorganism' exists. There are also implications for the many attempts to find salivary microbial and biochemical biomarkers that might be used clinically to assess caries risk. In the face of the multiplicity of microorganisms, this is nonsense! There are better ways to spend time in the clinic in order to control dental caries. This will be returned to later in the book.

The fact that the oral cavity is stuffed with bacteria does not mean that they need to be eradicated. They comprise part of the individual and each patient has his/hers own endogenous flora, but as a health professional one should always maintain correct personal hygiene (wash hands, use gloves, do not touch the dental chair/lamp, etc., with spit on the fingers) to avoid spreading infections between patients.

Microorganisms attach to all surfaces that are covered with a proteinaceous film and stick to them. In the oral cavity, however, the squamous epithelial cells desquamate and, hence, mucosal surfaces, such as cheeks, lips, and palate, do not allow growth of a biofilm, whereas tooth surfaces are stable. Wherever there is a stagnation area (between teeth, dental grooves, and fissures, along the gingival crevice, along filling margins, orthodontic brackets, dentures, etc.), a biofilm will form.

A dental biofilm is defined as a microbial community growing on a tooth surface. It is easily disclosed when the mouth is rinsed with a disclosing solution (Figures 2.1 and 2.2). On a clean surface, single cells (cocci) attach to the pellicle (the proteinaceous saliva film) within 12 hours and the cells start to multiply and form microcolonies within 24 hours. At this stage, the surface may feel matt or rough when moving the tongue tip over the surface of the teeth. If left undisturbed there is a microbial succession, continued growth and an increased species diversity resulting in a 'mature' or climax type of biofilm within a week. This structure is often referred to as dental plaque. Thickness

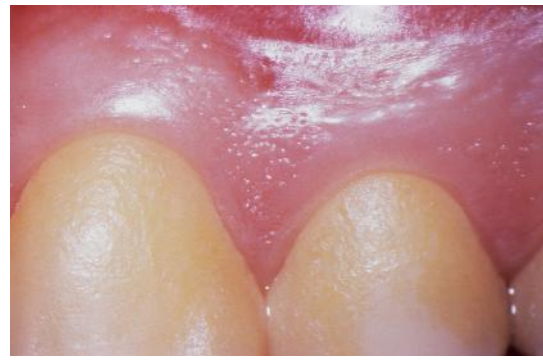

Figure 2.1 Cervical half of two teeth demonstrating a 24-hour dental biofilm.

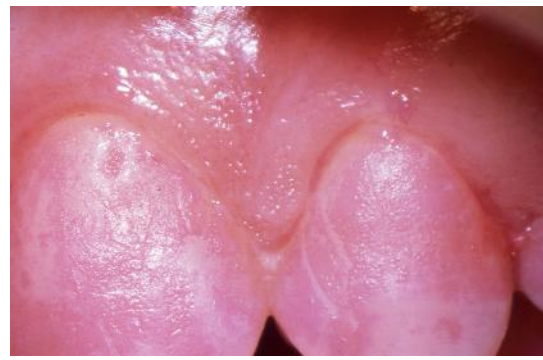

Figure 2.2 After application of a disclosing solution, the dental biofilm is more easily discernible.

and composition varies extensively between and within individuals. Some are rapid plaque formers—the dental plaque in elderly people who ignore oral hygiene can grow to a substantial thickness (Figure 2.3a,b). Rapid plaque formers require particular attention from a caries control point of view!

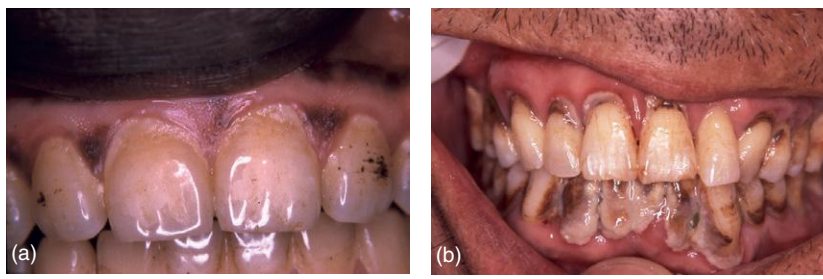

Figure 2.3 Dental biofilms of unknown age in an (a) African adult and in an (b) elderly Chinese. It is obvious that these patients do not use oral hygiene measures. Note how much difference there can be between individuals—and yet we talk about dental biofilm (or plaque) in general terms. Compare with Figure 2.1.

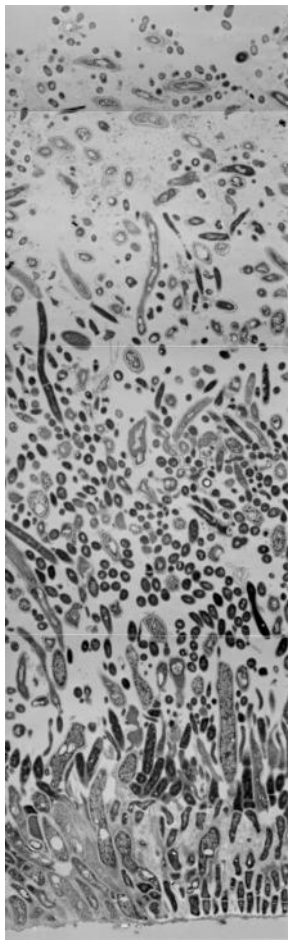

Figure 2.4 Transmission electron microscope picture of a dental biofilm after 48 hours Note the very loose structure composed of mainly cocci after 48 hours.

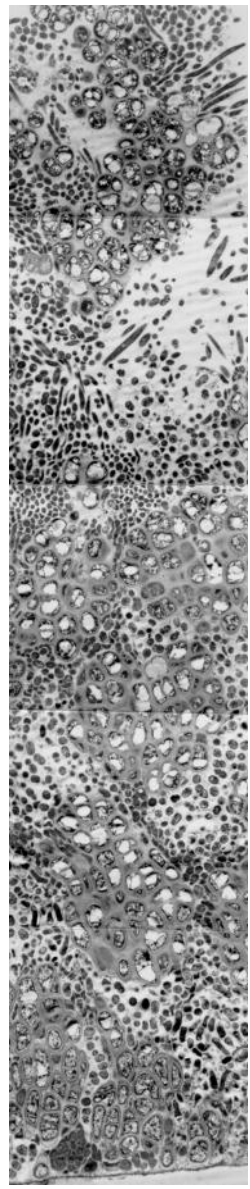

Figure 2.5 Transmission electron microscope picture of a mature biofilm after 2 weeks composed of many microcolonies of different bacteria surrounded by an intermicrobial matrix of highly varying density. Such a dental biofilm is not very accessible for various agents used, for example, in rinsing solutions. It is hopefully now appreciated why it is mandatory to mechanically disturb/partly remove such biofilms regularly as part of caries control.

You should be aware of the appearance of a dental biofilm in an electron microscope (Figures 2.4 and 2.5). It is highly diverse in composition with numerous microcolonies of different species. Close to the tooth surface the packing of microcolonies is very dense, with many of the colonies separated by channels of intermicrobial matrices. Such protein matrices are partly formed by

the microorganisms and partly derived from saliva. They vary in composition and texture, and the whole biofilm constitutes a complex viable mass, which is difficult to access and diffuse into. The surface of a dental biofilm is much more open in structure with new cocci constantly attaching and detaching.

It will be appreciated that this biomass behaves very differently to a test tube containing free-floating individual bacteria, but unfortunately most bacteriological studies have been conducted in test tubes or on agar plates where individual colonies of a single bacteria have been studied. These results may not be clinically relevant. An interesting example of this is that many antimicrobials have an effect on free-floating, test tube microorganisms (they are said to be in a planktonic stage), and inhibit or even kill microorganisms grown in culture. Chlorhexidine is a good example. However, once the microorganisms are established in a biofilm in the mouth, the antimicrobial agent cannot diffuse into the biofilm and any effect may only be observed on the superficial layers. This is why chlorhexidine as mouth wash or rinse has a very limited effect, even after repeated exposures (later in the book, when and how chlorhexidine may be used in certain patients will be described). In toothpastes it did not show an effect in controlled clinical trials (CCT) on caries incidence over time. Naturally, laboratory attempts have been made to mimic biofilm formation in a salivary environment, but it is all a surrogate of the complex situation in the oral cavity in vivo! Results from such laboratory studies are not necessarily transferable to the clinical situation.

You should also be aware that the deeper, consolidated layers of a dental biofilm can be difficult to remove during traditional toothbrushing. Saliva, particularly at the opening of the major salivary glands into the oral cavity, is supersaturated with respect to calcium and phosphate. Because of this, part of biofilm close to the opening of the salivary glands (buccal to the upper first and second molars and lingual to the lower incisors) may become mineralized as dental calculus. This may happen even in biofilms that are a few weeks old. Once formed, calculus adds to the difficulty of removing a biofilm with a toothbrush. This is one of the reasons oral health personnel remove calculus with instruments—it is done to facilitate cleaning by the patient.

2.1.1 Metabolism in the dental biofilm

The biofilm is characterized by continued microbial activity. The microorganisms metabolize salivary proteins and glycoproteins. These act as the primary source of carbohydrates, peptides, and amino acids for microbial growth. Different types of acids are produced very slowly and in small amounts from this metabolism, and these metabolic events result in continuous, minute pH fluctuations (see schematic illustration in Figure 2.6). The metabolism may be dramatically enhanced by changing the nutritional conditions, e.g. by adding dietary carbohydrates in excess such

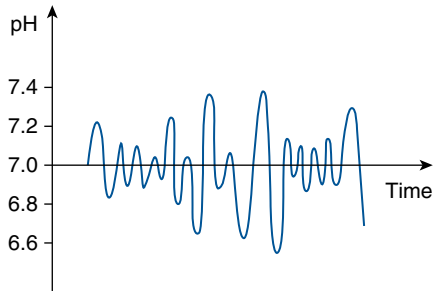

Figure 2.6 Schematic drawing indicating how metabolism in dental biofilms is reflected in numerous minute pH fluctuations throughout the day without adding sugars and meals.

as glucose, fructose, and sucrose. The outcome of the metabolism can be recorded as distinct pH fluctuations. In Figure 2.7, classical examples of such measurements known as Stephan curves are shown to correspond to lesions clinically characterized prior to recording. A Stephan curve is a pH curve measured regularly for 30 minutes in dental plaque after a rinse with 2% sucrose for 1 or 2 minutes. It is important to understand that this acid is not produced because the microorganisms wish to dissolve the dental hard tissues! The acid is produced because the microorganisms wish to survive. Excess sugar can kill bacteria (sugar kill) and to avoid this they have to metabolize the fermentable carbohydrates as rapidly as possible!

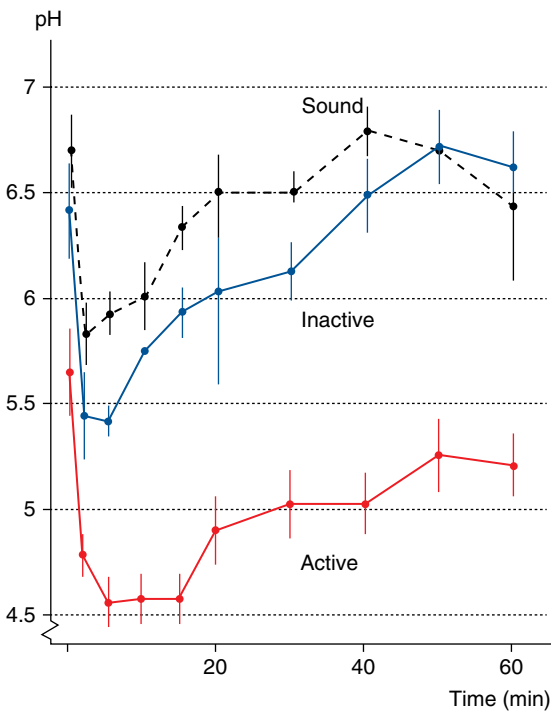

Figure 2.7 Stephan pH response curves measured in occlusal surfaces that prior to rinsing with sucrose were classified as either sound, exhibiting an inactive caries lesion or being classified as active lesions. Note the remarkable differences in the dental biofilm metabolism between these different surfaces reflecting the difference in ecologies of the biofilms!

Any shift in pH will influence the chemical composition of the biofilm fluid and the relative degree of saturation of this fluid with respect to the minerals is important for maintaining the chemical composition of the tooth surface. From the very moment of eruption into the oral cavity, the tooth surface apatite will continue to be subject to these chemical modifications on innumerable occasions. Most of these modifications are so subtle that they can only be recorded at nano-level. Surfaces that are frequently covered by biofilm (such as, for example, a cervical enamel surface) will gradually accumulate fluoride as a result of these processes in the very surface layers (outermost 100 μ), but such changes can only be recorded after years of exposure unless a subsurface caries lesion has developed. This will be explained chemically in relation to how a lesion develops later in this chapter.

Thus, the enamel surface is in a state of dynamic equilibrium with its surrounding environment. **When the cumulative result of the numerous pH fluctuations over months or years is a net loss of calcium and phosphate, of an extent that makes the enamel sufficiently porous to be seen in the clinic, it may be diagnosed as 'a white spot lesion'**. It is important to appreciate, however, that although the metabolic events may result in detectable caries lesion formation, most sequences of metabolic events tend to cancel each other, which is why the metabolic events should be considered intrinsic to biofilm physiology. The caries lesions arise when there is a drift in the metabolic events, that is when the pH drops result in a net loss of mineral. Thus, **dental caries lesions are a result of an imbalance in physiological equilibrium between tooth mineral and biofilm fluid**. These lesions are a symptom of biofilm activity, they reflect it, and this has a very important implication. To affect a lesion, concentrate on the biofilm, not the lesion! Next, how caries lesions develop is described.

2.2 Caries lesion development and progression

Under physiological conditions (pH 7.4) saliva and the oral fluids are supersaturated with respect to hydroxyapatite and fluorapatite. If this was not the case, the teeth would gradually dissolve, so this is the necessary precondition for the maintenance of tooth mineral in the mouth. In general, the higher the supersaturation with respect to the actual salt, the greater is the tendency for its formation. When salivary secretion is stimulated, this supersaturation is enhanced so most supragingival calculus consists of mixed fluorhydroxyapatite and hydroxyapatite. Occasionally, other calcium phosphates, such as octacalcium phosphate or brushite, are also a component of calculus.

An important clinical consequence of this knowledge is that there is no rationale in adding extra calcium or phosphate in various dietary compounds or tooth pastes/ mouth rinses – saliva is already supersaturated. The only effect might be enhanced formation of dental calculus!

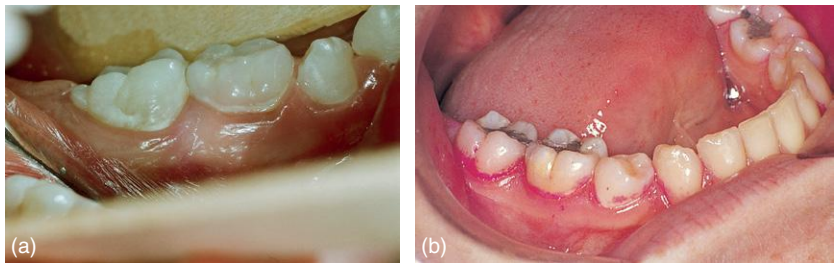

Figure 2.8 Clinical examples of white spot lesions. Note shape and location in stagnation areas. In 2.8b the lesions have been disclosed so they can be shown clearly to the patient before giving help with brushing.

When the pH in the dental biofilm decreases, the solubility of the tooth mineral apatite increases, **dramatically**. As a rule of thumb, the solubility of apatite increases by a factor of 10 with a drop of each single pH unit! Now you understand why the mineral of the tooth is vulnerable to an acidic environment.

Exposure to acids may lead to two types of lesion, **the caries lesion** (Figure 2.8a,b) and **erosion** (Figure 2.9a,b). The initial stages of carious lesion formation are characterized by a partial dissolution of the outermost enamel, almost an erosion.

As pH is lowered in the oral fluids, saliva and plaque fluid, the supersaturation with respect to hydroxyapatite is reduced and at 'critical' pH (Figure 2.10) the fluids become saturated with respect to hydroxyapatite. Because fluorapatite is less soluble than is hydroxyapatite, plaque fluid remains supersaturated with respect to fluorapatite when it is undersaturated with respect to hydroxyapatite. Under these conditions a carious lesion forms. Subsurface hydroxyapatite is dissolved, while fluorhydroxyapatite is formed in the surface layers of enamel (Figure 2.11). In general, the more undersaturated the plaque fluid, with respect

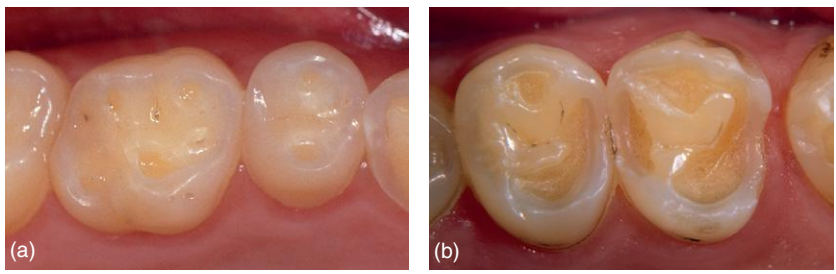

Figure 2.9 Eroded occlusal surfaces exhibiting different degrees of erosion, starting with a rounding of contours to almost total exposure of the dentine after removal by the acids of the covering enamel. (a) Molar and premolar with a little wear. (b) Premolars with deep wear down to the yellow dentine.

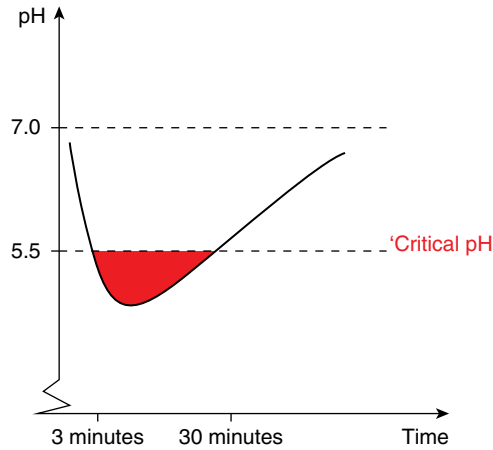

Figure 2.10 Schematic drawing of a Stephan response curve marking the location of the so-called critical pH.

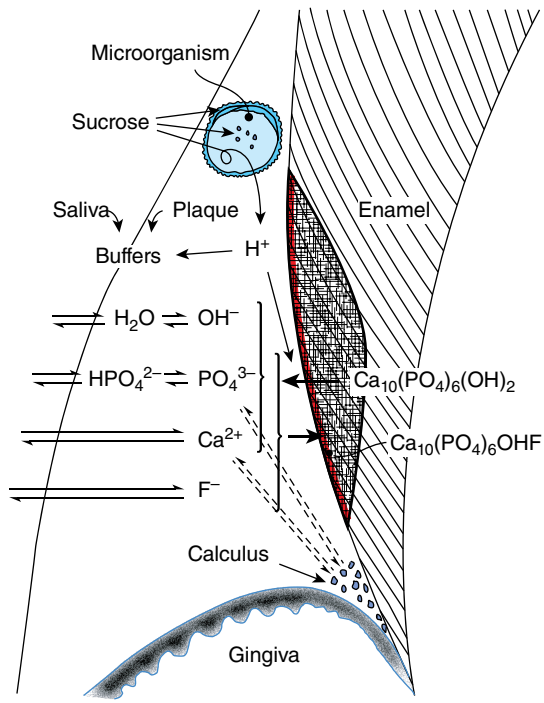

Figure 2.11 Drawing indicating the principal chemical events that lead to a subsurface enamel caries lesion. In the pH range of 5.5–4.5, saliva will be undersaturated with respect to hydroxyapatite, while supersaturated with respect to fluoridated apatite. Therefore, hydroxyapatite will dissolve from the enamel interior while fluoridated apatite will deposit in the surface enamel. The end result is the subsurface enamel caries lesion.

= Surface Zone

to hydroxyapatite (i.e. the lower the pH), the greater the tendency for dissolution of the enamel apatite.

However, the concurrent supersaturation with respect to fluorapatite is responsible for the maintenance and integrity of the surface layer. The more supersaturated the solution, with respect to fluorapatite, the thicker and less demineralized the surface layer remains. When sections are cut through an active, so-called white spot lesion, and examined, either by light microscopy or by microradiography, it is apparent that the loss of mineral occurs in the subsurface enamel and that the thickness of the surface zone may vary considerably (Figures 2.12 and 2.13). When the enamel surface is examined in a scanning electron microscope, active enamel caries surfaces appear moth-eaten with partial dissolution of crystals so that there is an enhanced spacing between crystals (Figure 2.14). (Remember, clinically, such surfaces appear matt, with loss of lustre, and when the tip of the probe is gently moved across the surface there will be a feeling of roughness).

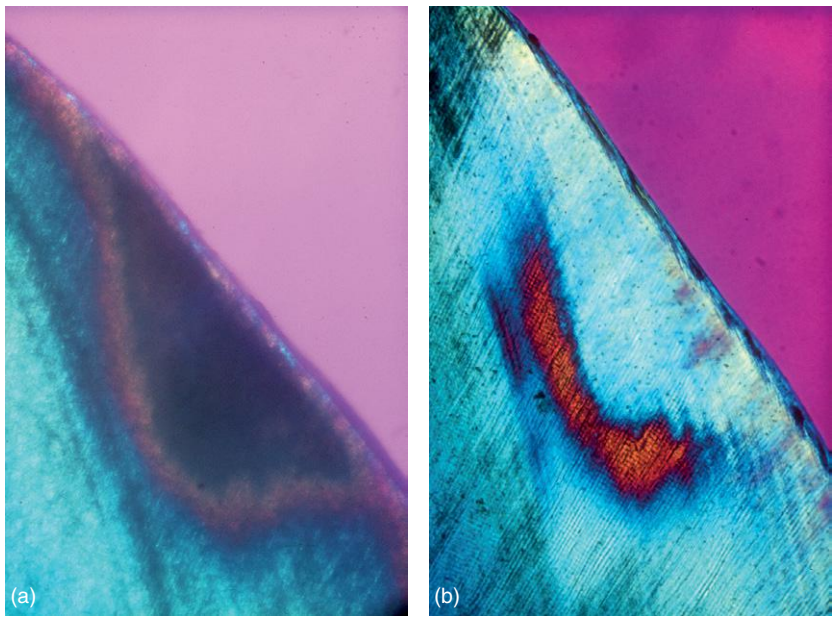

Figure 2.12 When sections of teeth are examined in water with polarized light the pores in the enamel are filled with water. Water has a lower refractive index than enamel. The white spot caries lesion has a pore volume of more than 5% on a smooth surface (a). When the same section is examined in a solution which has the same refractive index as enamel, but a molecular size that does not allow it to sieve into the small pores at the advancing front of a lesion, a so-called dark zone is observed (b).

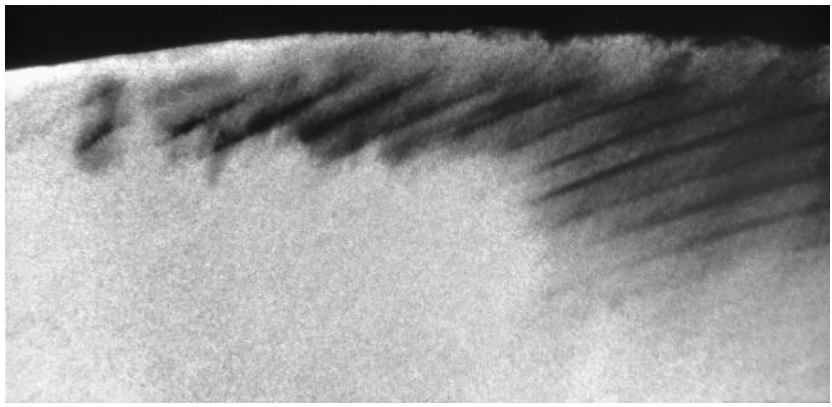

Figure 2.13 When sections are examined by X-rays (microradiography) the calcium loss is directly shown as darker areas in the otherwise white, highly mineralized enamel. Note how the surface enamel varies in mineral content, but is better mineralized than the body of the lesion.

It is very important to appreciate, however, that in a cariogenic oral environment with innumerable fluctuations in pH for days, months, and years, these surface zones will be constantly dissolving and re-depositing mineral. The formation of fluorapatite at the expense of hydroxyapatite in surface enamel leads in time to a higher content of fluorhydroxyapatite in the surface layer of the

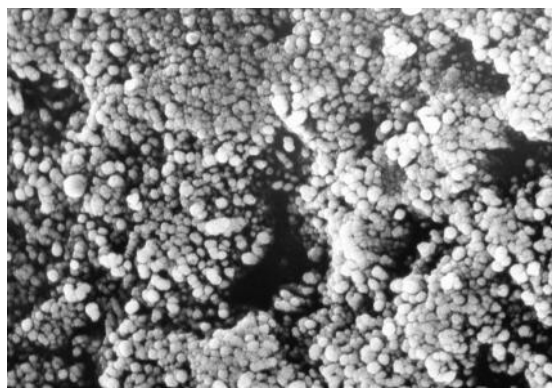

Figure 2.14 When examining the enamel surface of an active white spot caries lesion in the scanning electron microscope (SEM), however, the surface appears moth eaten because the spaces between the individual crystals are widened. It is porous and a probe tip will feel the roughness when gently moved across the lesion.

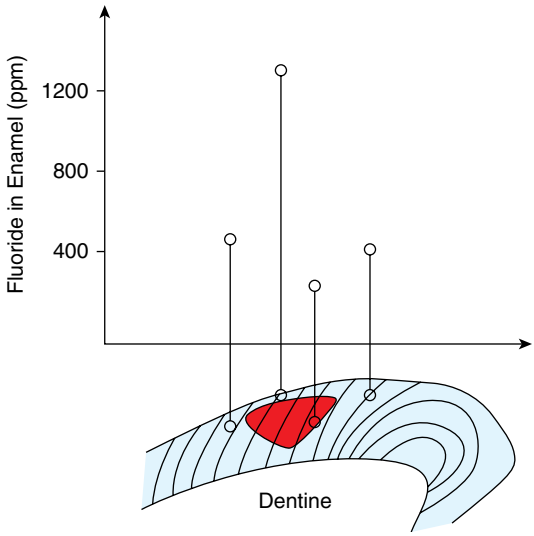

Figure 2.15 Schematic drawing showing the variation in fluoride content in a white spot caries lesion. Note that the highest content is in the surface zone covering the body of the lesion! The explanation is evident when comparing with Figure 2.11.

Data from *Caries Research*, 11, 2006, Weatherell, J. A., et al, 'Assimilation of fluoride by enamel throughout the life of the tooth', pp. 85–115.

carious lesion (Figure 2.15). So, the fluoride concentration in the subsurface body of the lesion of the enamel is not increased. As long as the surface layer remains with a reasonable mineral content, fluoride is not taken up into the body of the lesion, but an enamel surface covering a white spot lesion contains more fluoride than the surrounding sound, normal enamel! In a number of respects the surface layer exerts a protective effect to prevent further dissolution of the lesion body as long as the pH fluctuations are in the range of 4.0–5.6.

If the caries challenge is maintained, these processes continue and the dissolution of the mineral continues steadily because the acids diffuse into the deeper parts of the enamel along the naturally occurring pathways, namely the rod (or prism) boundaries. Therefore, in microscopy of sections with caries lesions it can be seen that the increased porosity enhances the appearance of the rod structure in the enamel. It is also evident that in smooth surfaces the diffusion of acids is most pronounced, corresponding to where the biofilm covering the lesion is thickest so in the microscope the shape of a classical lesion is triangular (Figure 2.12). However, in three dimensions it is actually cone-shaped with the base of the cone at the enamel surface.

If the lesion is in an occlusal surface with grooves and fissures, the spread of acids similarly follows the rod pattern. Therefore, around the sides of a fissure the rod patterns will result in a pattern of spread seen as triangles in two dimensions on a slide on either side of the fissure. When the demineralization reaches the dentine the further spread of demineralization is as described for smooth

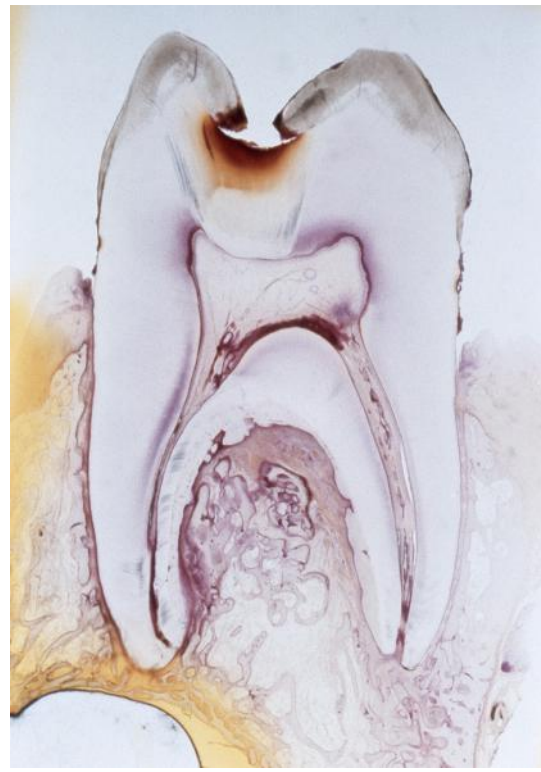

Figure 2.16 In occlusal surfaces a caries lesion starts at each side of a fissure and, because of the anatomy, the mineral loss occurs in the enamel following the prism direction. This creates a cone shape with the broad base towards the interior of a tooth. Therefore, when the enamel breaks down, the cavity appears bigger than assumed at the surface because the dentine is involved at the base of the cone.

surfaces, but because it happens around a groove or fissure in three dimensions, it takes the shape of a large cone with the base towards the interior of the tooth (Figure 2.16). Once such a lesion has reached a point where the enamel breaks apart, the resulting cavity will appear larger at the base of the cone, giving the clinician the feeling that occlusal caries is undermining the enamel. Another important consequence is that when such cavities are opened with a bur they appear much larger than the immediate size of the opening of the cavity detected clinically at the surface (compare with Figure 2.17a,b).

Returning to the spread of mineral dissolution in the smooth surface lesion, in almost all cases the spread of demineralization is a slow process, which may take years even if no caries control is attempted. Therefore, at the apex of the lesion the dissolution may reach the enamel–dentine border and continue into dentine (Figure 2.18). Now the pattern of spread is determined by the structures of the dentine. The demineralization will appear most pronounced corresponding to the tip of the enamel lesion, but it is important to notice that there are signs of dissolution along the dentine–enamel border corresponding

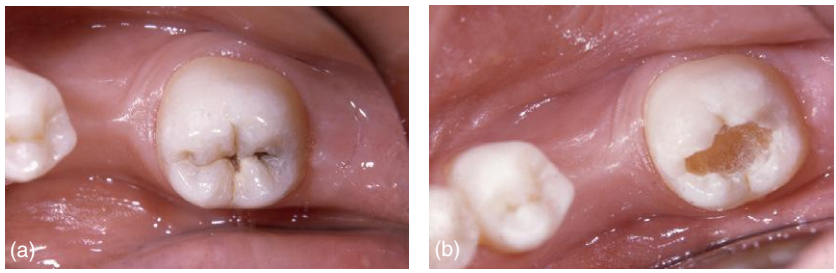

Figure 2.17 The description of Figure 2.16 is clinically reflected when comparing the occlusal lesion (a) before and (b) after opening the cavity in this lower second molar.

to where the entire enamel rods at the surface of the enamel are affected. Examination of histological sections may create an erroneous impression that the dissolution spreads along the dentine–enamel surface, whereas it is a simple reflection of the total enamel area affected at the tooth surface. As the dental biofilm becomes thinner towards the periphery of an affected area, the magnitude

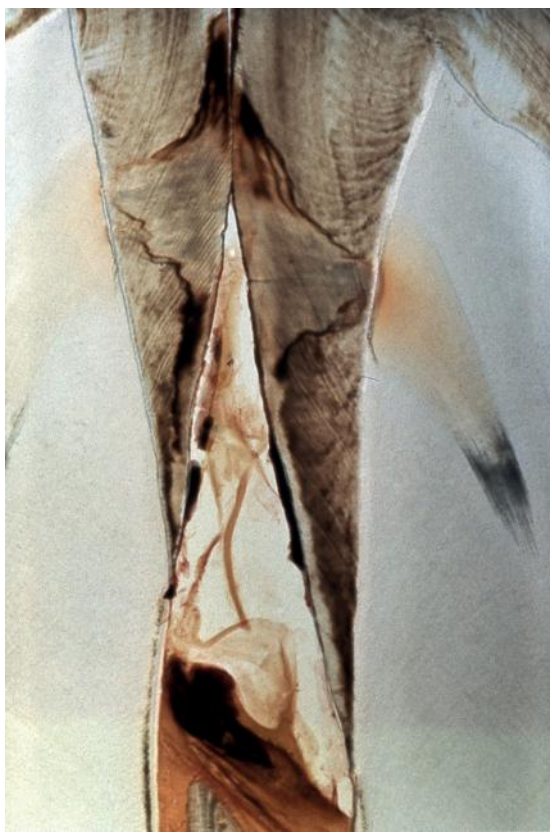

Figure 2.18 A section of an approximal space between two neighbouring molars with two caries lesions extending into dentine, but without cavity formation. Note the dentine reactions involve all the dentinal tubules affected by the acid attack at the enamel surface.

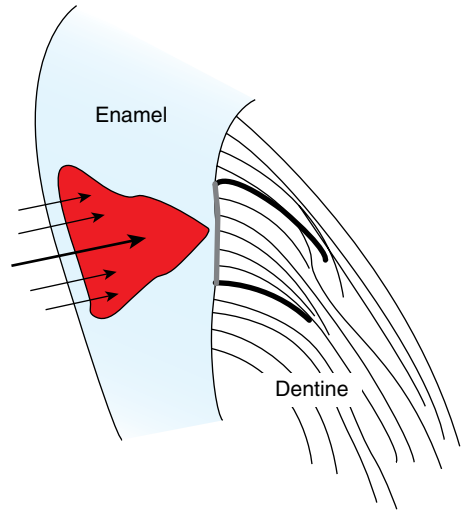

Figure 2.19 A drawing that, in a simplified way, shows how the intensity (indicated by the relative size of the arrows) of acid fluctuations in the dental biofilm at the enamel surface creates the typical triangular shape of the enamel lesion.

of demineralization is less and, naturally, the corresponding distant effect on underlying dentine is less. In the schematic drawing (Figure 2.19) this principle is indicated by the thickness of the arrows.

In bitewing radiographs, such lesions may reflect themselves as shadows extending partly or totally through the enamel (Figure 2.20). Based on such an image the clinician may decide to drill and fill but this would be **totally incorrect** because these lesions are rarely cavitated and can be controlled without restoration. A careful clinical examination of these lesions will often confirm this lack of cavitation. The consequences of this overzealous attitude to the prescription of fillings can be seen in a population of Danish 12 year–old school children in Chapter 8.

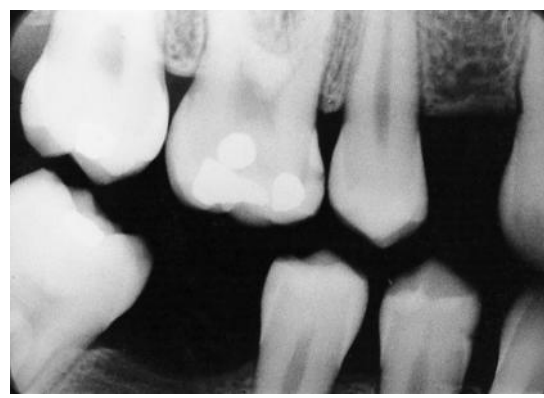

Figure 2.20 A bitewing radiograph showing caries in enamel and dentine on the mesial aspect of the upper first molar. A lesion is also visible on the mesial aspect of the lower first premolar.

When about 30–40% of the mineral in the enamel lesion is lost, it becomes so porous and fragile that it may easily break apart and a cavity forms (Figure 3.7). Mechanical stress on the enamel favours this and, if a dentist starts sticking a probe into such a surface, it will inevitably result in a cavity (see Figure 3.5a,b), which opens up the deeper enamel where a thick microbial mass will form with enhanced diffusion of acids deeper into the tissues. Before dealing with what happens in caries-affected dentine what is meant by remineralization of caries lesions will be considered.

2.2.1 Remineralization of enamel

Remineralization will inevitably be preceded by a demineralization. Occasionally, in the clinic it can be observed how a typical 'white spot lesion' with time becomes less opaque and even smaller in surface area (Figures 2.21 and 2.22). However, this is a fairly rare situation. Most often a white spot lesion remains as a scar in the tooth. **This implies that it will only be possible to reverse a lesion by starting the caries control measures while the lesion is in an active stage**. That is why assessment of lesion activity is so important when diagnosing lesions! Once a lesion has become inactive (or arrested) its appearance cannot be changed unless the surface is mechanically polished or ground or by stripping a layer off it with a strong acid. These procedures strip off the surface zone of the lesion.

In order to understand this it is necessary to return to the microscopic appearance of the enamel surface during dissolution. Only when the crystals are partly dissolved are they able, with time, to allow calcium and phosphate ions to redeposit on the partly dissolved crystal surfaces, and in the interior of single crystals.

Remineralization of enamel subsurface caries lesions requires that calcium and phosphate ions are able to diffuse into the porous subsurface enamel usually through the relatively intact surface zone. All pores in enamel – whether in normal enamel or porous carious enamel – are not 'empty', but filled with water and proteins. In the surface of the enamel salivary proteins penetrate, but it should be acknowledged that in vital teeth there is an outflow of ions from dentine through the enamel due to the blood pressure in the pulp. Ions in general only slowly penetrate into a lesion with an intact surface layer, even under extreme laboratory conditions. A single pH fluctuation in plaque is unlikely to affect the pH in the interior of a lesion. Remineralization of dental lesions requires the presence of partially demineralized apatite crystals that can grow in size as a result of exposure to solutions supersaturated with respect to apatite. The formation of entirely new crystals in a subsurface lesion is not likely, whereas it is seen in the surface of the enamel lesions where numerous pH fluctuations have occurred. These conditions set limits to what can be expected from remineralization.

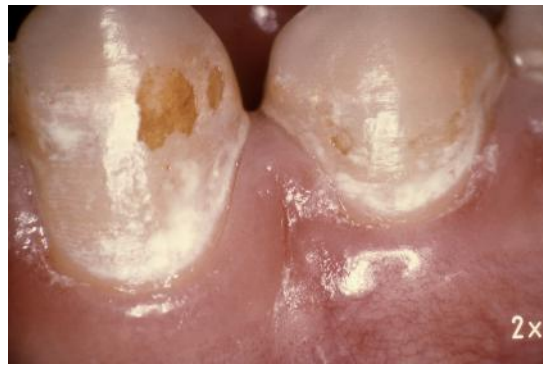

Figure 2.21 Extensive white spot caries lesions at the active stage.

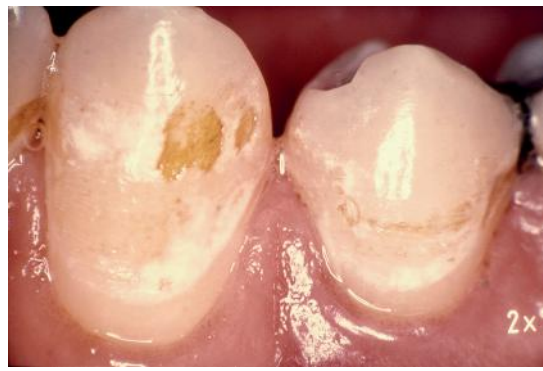

Figure 2.22 The same lesions as shown in the previous figure after weeks of intensive polishing with a fluoride-containing paste and daily brushing with fluoridated toothpaste. This reflects the maximum possible effect of so-called remineralization. The surface areas of the lesions have diminished a little. As a result of good oral hygiene the gingival inflammation has disappeared and, as a consequence, the marginal gingiva has retracted exposing a cervical rim of sound enamel cervical to the inactive lesions, which are now left as scars in the enamel.

Please be aware that often there are claims of new remineralizing solutions, but these claims are often based on very artificial laboratory experiments. What happens in the oral cavity is an entirely different matter!

Briefly it is necessary to return to erosions. In contrast to dental caries, which is a result of acid production in a dental biofilm (and hence 'site specific' to regions of biofilm stagnation at tooth surfaces), erosions are a result of the removal of thin layers of surface enamel when exposed to extrinsic acids at low pH (most often from acidic foods and soft drinks, regurgitated stomach acid, or occupational

exposure to airborne acids) and involves buccal/occlusal and/or palatal surfaces throughout the mouth. The removal of enamel layers is often exacerbated by abrasion and attrition, and even the tongue being frequently moved over the lingual surfaces of the teeth can add to removal of softened enamel surfaces.

Since erosion is a lesion in which enamel is etched away in layers and the crystals are lost, remineralization of eroded enamel cannot be expected, despite such surfaces being exposed to supersaturated saliva for long periods. The etched surface is covered by the salivary proteins and the etched outermost crystals are rapidly abraded away.

In contrast, the active carious lesion contains partially demineralized crystals and considerable remineralization of surface enamel in lesions free of plaque has been observed, in particular in caries lesions developed during orthodontic treatment. So, what is dealt with clinically when talking about remineralization is predominantly **surface changes**, which are a combination of abrasion of the porous enamel, and a slow re-deposition of mineral in and onto the partly dissolved crystals (Figure 2.23). Owing to slow diffusion, however, it does not seem possible to maintain the necessary supersaturation in the lesion fluid and, therefore, remineralization of the lesion body is not obtained to any significant degree in vivo.

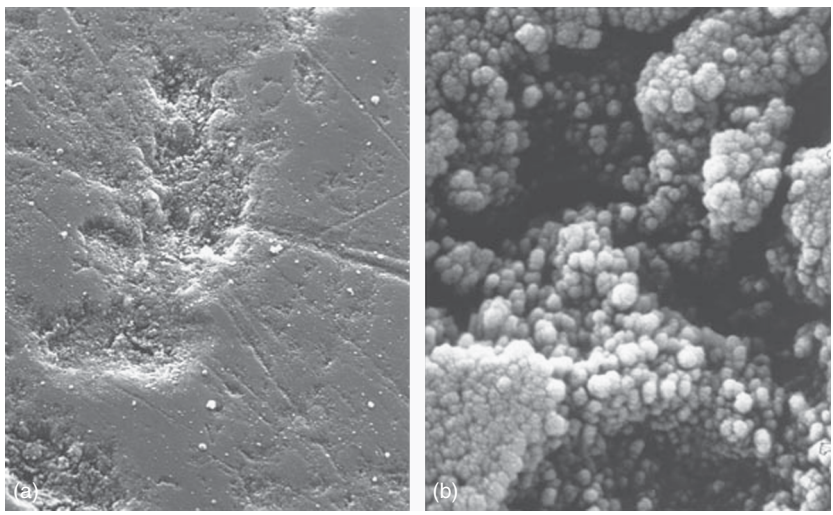

Figure 2.23 Scanning electron microscope pictures of surface enamel of an inactive caries lesion. Note how the surface appears polished, leaving small irregularities where crystals can be discerned. (b) At the higher magnification of part of the small irregularity, it is evident that where the polishing cannot affect the bottom of these irregularities, the spaces between the crystals in inactive lesions have been reduced. Compare with the SEM of surface enamel of an active caries lesion in Figure 2.14.

The surface layer of the lesion protects the underlying lesion body not only from demineralization, but also from remineralization. Remember that deposition of ions onto and occasionally into partly dissolved crystals is an extremely slow process. It may take months or rather years to observe a clinical effect.

2.3 Progression of caries lesions in dentine

As already stressed, dentine demineralization can be seen in histological sections long before there is any cavity formed in the enamel and it is stressed that the basic structure of dentine determines how further spread occurs. Basically, the dentinal tubules determine the direction of the reactions (Figures 2.18 and 2.19). The zone of demineralization gradually spreads toward the pulp, but is most often preceded by a zone of **translucent dentine**, which reflects an attempt from the vital pulp and odontoblasts to arrest the process by partly or totally occluding the dentine tubules with minerals. This reaction is called tubular mineralization or 'sclerosis'. It is very likely that the consequence of the mineralization is a decreased permeability of the tissue. It is particularly remarkable that also along the periphery of a dentine lesion a thin zone of tubules also show an enhanced mineral uptake (Figure 2.24). These tubules are involved from the sides of the tubule, which probably explains why the zone is much thinner than the zone at the advancing front of the demineralization.

In the pulp corresponding to the odontoblasts involved, a deposition of irregular **tertiary dentine** is usually observed (Figure 2.25). Once again, this reaction decreases permeability and is probably a defence, attempting to wall off the pulp from the bacterial assault. Chapter 5 describes how these two defence reactions, tubular mineralization, and tertiary dentine, can be harnessed by the careful operative dentist.

Once a cavity is formed, the hole in the tooth is filled with large accumulations of retained microorganisms and biofilm. This teams with microorganisms, and

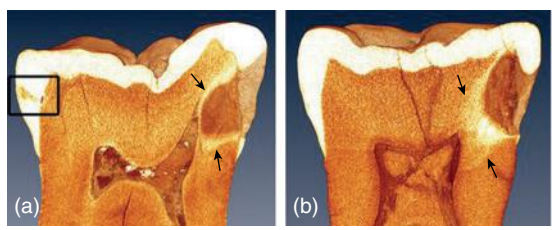

Figure 2.24 A micro-computer tomography (micro-X-ray method) scan through a 2000-year-old Roman tooth (see Figure 2.32) with approximal lesions (a non-cavitated lesion in the framed site in (a)) and a large cavitated lesion in (a) and (b). Note the mineral deposition walling-off the cavity along the borders of the dentine cavity (marked with arrows).

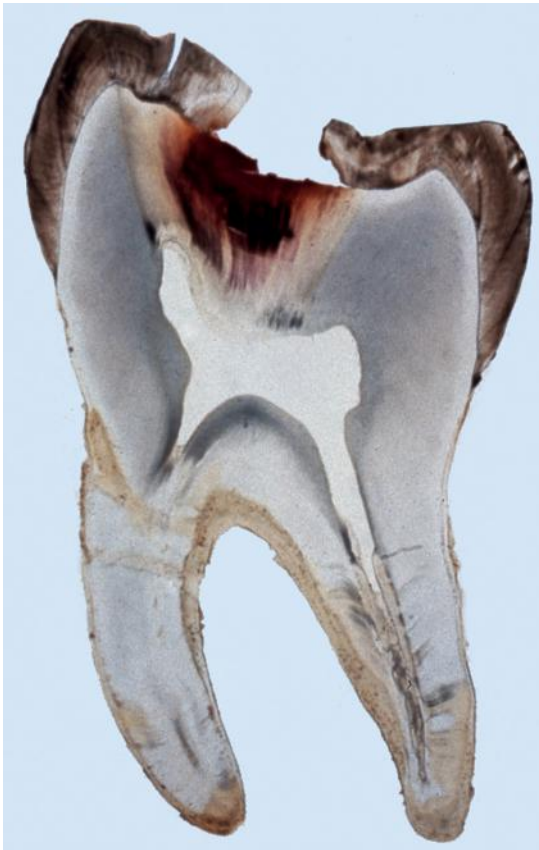

Figure 2.25 Histological ground section through an undemineralized molar with a large occlusal active caries lesion. Note that the lesion almost extends to the pulp chamber and along the advancing front there is a rim of tertiary dentine. It is also remarkable that, although extensively demineralized, the gross structure of the dentine remains. Assuming that the pulp is vital, imagine what would happen if you choose to drill – or perform caries control where the softened dentine is partly removed, and partly remineralized. Compare this with the clinical picture in Chapter 3 (Figure 3.10) of an inactive deep occlusal cavity in a deciduous molar!

they penetrate into the tissue. When reaching dentine, in addition to continued acid production, they also start a proteolytic activity. The consequence of this is that the partly demineralized collagen in the dentine becomes degraded. When probing into a cavity this is the soft, sticky, yellowish/brownish carious dentine (see Figure 5.6a), which is commonly removed when excavating a lesion.

Of course, microorganisms penetrate the individual dentinal tubules that are partly demineralized, but probably these advancing bacteria are of limited importance as it is the bulk of microbial mass in a cavity (sometimes called the biomass) that is the main acid-producing fabric. This may be a very important message from a clinical point of view as we shall come back to in Chapter 5.

The pulp is a vascular connective tissue with highly specialized cells, the odontoblasts, producing the dentine during tooth development. As the cells produce a collagenous matrix, which gradually mineralizes (much smaller crystals

than enamel crystals—in dentine they are the same size as in bone), the cells withdraw leaving a cellular extension (the odontoblast process) located in the dentinal tubules. In the erupted tooth the pulp tissue (with the odontoblasts located along the inner dentinal wall) is totally surrounded by hard tissue except for where the vascular supply to the tissue enters at the root apex. In other words, the dentine is part of a **vital** tissue in contrast to erupted enamel. Any external injury (chemical, thermal, mechanical, etc.) to the surface of the tooth may result in cellular reaction deep within the pulp and eventually cause pain.

Physiological abrasion of teeth, which removes substantial amounts of enamel at the cusps, with time, provokes the formation of additional dentine corresponding to the odontoblasts involved. When the gums retract and expose root surfaces, these may be painful whenever the patient drinks/eats cold and hot items such as cold water, ice cream, and hot tea. This is a simple reflection that the pulpodentinal tissue is alive because the odontoblastic processes extend almost to the surface of the outer dentine, and the tissue fluid that surrounds them is sending 'chemical signals' to the cells and the nerve endings in the pulp tissue.

Now one will appreciate why pH fluctuations, even at the surface of the partly demineralized enamel, may provoke a pain response from the tooth. Just feed sugar to a child with caries lesions and the pain reactions will be noticed. In fact, the pulp tissue is reacting because there is an inflammatory reaction of varying intensity in the connective tissue of the pulp. This inflammation is just a fundamental process that can be seen in all connective tissues in the body if they are injured; sunburn, for example, or too hot water on the skin, produces a red area and this is inflammation as a result of injury. Part of the inflammatory response is an increase in blood flow (the affected area swells and gets reddish). In a finger, the swelling may be painful when the swelling is substantial, but in the pulp such a vascular reaction will cause pain because swelling is not possible as the tissue is trapped inside the dentine.

When dealing with active caries lesions pain may often be provoked, but as long as it rapidly disappears once the stimulus (hot, cold, sweet) is removed, it is a **reversible pulpitis**. In patients with open cavities the pain symptoms can be removed by the patient themselves if they are taught how to remove, with a toothbrush, the majority of the microbial masses in the cavities. Usually, it will take about 3–4 weeks to avoid the pain by this simple caries control measure, but for the patient it is a very useful way of suddenly appreciating that **they themselves** are able to diminish the pain. When it is no longer possible to eliminate or reduce the pain reaction quickly—it persist for longer periods and occurs spontaneously at night when the blood flow to the head is increased because of the horizontal position—the clinician talks about **irreversible pulpitis**. This differential

diagnosis is crucial because now, without endodontic treatment, the condition may gradually result in necrosis of the pulpal tissue and the inflammation may extend outside into the periapical tissues. Should these areas become infected the result is a **periapical abscess**. Now the patient experiences very severe throbbing pain. As long as the dental pulp does not show signs of irreversible changes, it is of paramount importance to do the utmost to control further caries progression even in large cavities in order to preserve the pulp. We return to this in Chapter 5.

2.4 How about the role of mutans streptococci in dental caries?

You will often see in the literature that mutans streptococci are particularly important for the development of dental caries and can even be used in various caries predictive models. Why is there this attempt to link a particular type of microorganisms to dental caries? Again, it is necessary to return to what shaped caries research in the last 50 years to fully appreciate how this narrow way of looking at caries microbiology arose.

Not all bacteria in the oral cavity are equally able to ferment carbohydrates. For this reason, it is logical to look for 'major caries pathogens'. Lactobacilli were identified as major acid producers and then, from the beginning of the 1960s, much focus was on mutans streptococci. They produce a particularly sticky extracellular polysaccharide and they are **acidogenic,** which means they are good acid producers. They are also **aciduric**, which means they can survive in acid environments. In fact, both lactobacilli and mutans streptococci prefer to grow and metabolize at low pH to the extent that they tend to overgrow in very acid conditions.

Also in the 1960s, hamster and later rat studies showed that if the animals were infected with certain streptococci and fed on a cariogenic diet (frequent sucrose every day) the rodents developed extensive fissure caries. This property apparently spread to the siblings born and kept in the same cages as their mothers, so it was claimed that dental caries was 'an infectious and transmittable disease'. The fact that rodent puppies eat their parents' faeces and get 'infected' in this way, was unfortunately ignored. However, this concept of an 'infectious and transmittable disease' came to dominate cariology when dealing with humans in the following decades. We now know that this is incorrect and to appreciate why the following facts should be considered:

- Mutans streptococci are part of the resident flora, but are in very low numbers in most caries free individuals.
- *Streptococcus mutans* is often found at higher concentrations in plaque covering white spot lesions.

- However, there is no association between approximal caries lesions and the presence/absence of *S. mutans* or the total mutans counts in a biofilm and the plaque pH response in children.
- Children with increased numbers of caries cavities exhibit a higher number of salivary mutans counts but subgrouping of children based on different levels salivary mutans counts does not allow a distinction between high or low caries experience in children.
- The number of *S. mutans* or lactobacilli in dental biofilms does not explain the variation in caries experience. Indeed, high number of these microorganisms may be found at sites without signs of demineralization.
- A dramatic decline in dental caries has been observed in well-conducted trials in populations followed over a short period of time, without apparent concomitant changes recorded in salivary mutans levels in the populations.
- There are many acid-producing microorganisms in addition to *Streptococcus mutans* and lactobacilli.
- A thorough review of the extensive literature concludes that **no single type of microorganism can be claimed to be the primary cause of either root or enamel caries**.

Thus, with the emerging appreciation of the role of dental biofilms, and their characteristics as described previously in this chapter, came a growing appreciation that the concept should not be to search for one organism, but to realize that the biofilm conditions at a particular site favour a particular mix of microorganisms specifically adapted to live at a that particular site. This is known as the **'ecological plaque hypothesis'**. It is a dynamic way of interpreting the role of dental biofilms.

Remember there is an intensive metabolism constantly going on in any dental biofilm resulting in minute pH fluctuations at the tooth surface. Acidogenic/aciduric microorganisms constitute a small proportion of most biofilms and, in an oral environment with a neutral pH, these microorganisms are weakly competitive and constitute only a minor fraction of the total microbial mass. Introducing mutans streptococci experimentally in such an ecosystem in physiological balance does not result in caries lesion development.

However, if the frequency of fermentable carbohydrate (sugar) intake increases from food, snacks, biscuits, soft drinks, changes in ecology of the biofilm at areas of the mouth begin to be observed, where biofilms are relatively protected and reach a stage of 'maturation', i.e. stagnant areas. Fermenting the carbohydrates enhances biofilm metabolism and significantly influences the ecology. This may be in the oral cavity in general or just in certain sites in the mouth. In this context remember that, despite the fact that the oral cavity is an open sink,

particular parts of the oral environment represents ecological niches where some sites, such as the dorsum of the tongue, lingual/palatal parts of the dental arches, behave differently from buccal/labial parts and approximal spaces. Sites where salivary ducts open into the oral cavity also present their own unique and protective environment.

The enhanced sugar metabolism will result in larger drops in pH in the biofilm and longer periods of lower pH at the tooth surface. This condition favours the proliferation of aciduric bacteria such as mutans streptococci, lactobacilli, and others. This results in more acid being produced and at faster rates, and a vicious circle develops resulting in a net loss of mineral in the surface of the tooth. **Thus, caries becomes a consequence of changes in the natural balance of the resident microflora as a result of an alteration in the local environment**.

You will now understand why the concept of 'caries being an infectious and transmittable disease' should be replaced by **'caries being a biofilm induced disease—the biofilm being composed of the resident microflora which is in a dynamic equilibrium with the oral environment'**. This could not be more important. The implication is that caries should be controlled by altering the biofilm by disturbing it regularly with toothpaste containing fluoride and a diet that is not packed with frequent sugar.

2.5 Root surface caries

Of course, this type of dental caries can only develop when the root surfaces become exposed to the oral environment with age (Figures 2.26 and 2.27). The basic physico-chemical conditions taking place are similar to those described for enamel caries. If the cementum is not scaled away as a result of vigorous scaling by hygienists or dentists, the early stage of a lesion appears as a surface

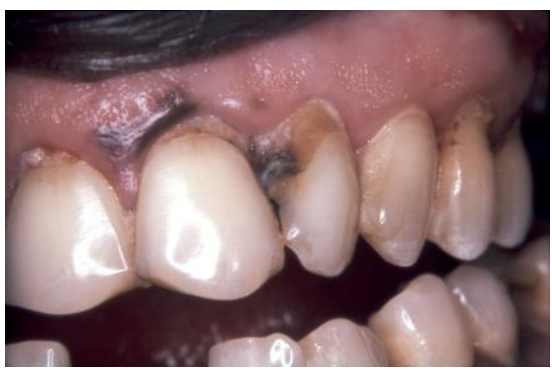

Figure 2.26 Root surface caries lesions may start along the cervical region when gingiva starts to retract. No probing is needed.

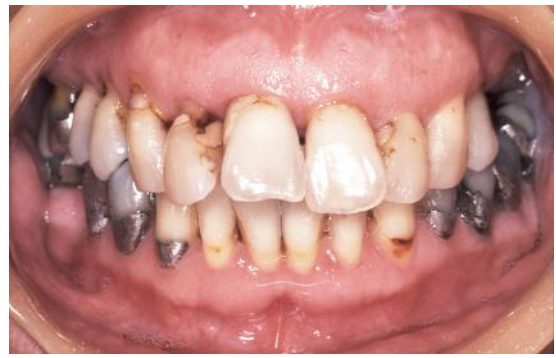

Figure 2.27 It is obvious that attempts to fill ongoing active root surface lesions are doomed to fail unless caries control measures are undertaken.

softening accompanied by a subsurface demineralization. As soon as the demineralization reaches the dentine the spread of lesions follow the same patterns as described earlier (Figure 2.28). Caries lesions on root surfaces are invaded early on by microorganisms from the dental biofilm. However, this does not preclude the possibility of arresting a root surface caries lesion—even accessible cavitated lesions. As soon as the covering microbial biofilm is mechanically removed (toothbrush and fluoridated toothpaste), the softened brownish yellow surface is gradually polished and starts to become darker. It may take many months before the lesion surface is hard and shiny and appears dark almost black (Figure 2.29).

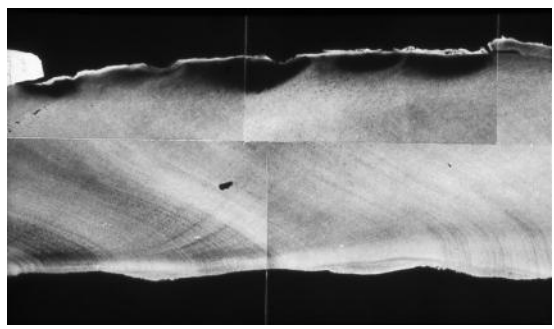

Figure 2.28 Microradiograph through root surface with subsurface caries lesions extending along the entire exposed root surface. Note the very thin rim of better mineralized dentine surface compared with the subsurface loss of much more mineral. It is also remarkable that, although the entire root surface is affected by caries, the lesions are not deep—again do not start to drill and fill such lesions, but instruct the patient to brush and thus twist these active caries lesions to become inactive. Compare the clinical results when looking at next figure.

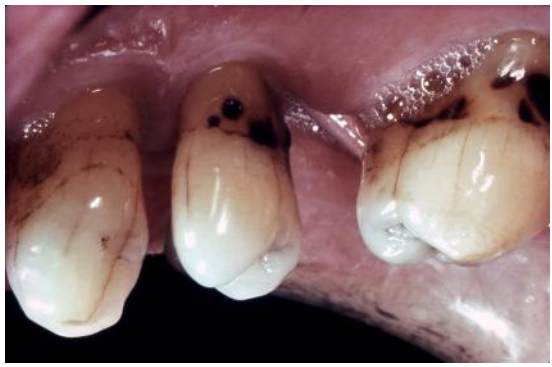

Figure 2.29 Inactive root surface caries lesions which are entirely dark and hard on probing. These are scar tissue resulting from previous active caries lesions! Imagine if a dentist had drilled and filled such lesions.

2.6 The role of saliva in caries development

Saliva plays a key role in oral health. It not only lubricates the dentition and keeps the oral cavity humid and moist, but it contains electrolytes and important proteins. Its role in caries development is best illustrated in patients with diminished (hyposalivation), or totally arrested saliva production. These patients are without doubt the most challenging to care for and this is returned to in Chapter 7. However, some basic knowledge about saliva is important.

Whole saliva is the mixed secretion produced by the three paired major (produce almost 90%) and numerous smaller salivary glands (produce only about 10%). It contains millions of bacteria, vast amounts of desquamated epithelial cells, but it is predominantly composed of water. The composition varies extensively and the flow rate varies as well. Thus, at night there is hardly any saliva secretion, which everyone who sleeps with an open mouth will know when waking up! The mouth is so dry it feels terrible, like a piece of sandpaper.

Salivary flow rate strongly influences the final composition of whole saliva. For example, the concentration of bicarbonate, an important buffer in saliva, varies depending on flow rate; the more stimulated the saliva production, the higher the levels of bicarbonate. The secreted bicarbonate is derived from carbon dioxide, which evaporates shortly after entering the oral cavity. Therefore, a particular tendency to calculus formation on buccal surfaces of first upper molars and lingual surfaces of lower incisors because salivary ducts open in these areas can be observed. Salivary pH is strongly dependent on the secretion rate. In healthy individuals, pH varies between 6.0 and 7.5, being most alkaline under stimulated flow rates.

Saliva is stimulated during the action of eating. How the mere smell of certain foods may result in the subjective feeling of saliva flowing down the cheeks is a familiar reaction– just think of a delicacy and it starts secreting. An important

role of saliva is to dilute and eliminate substances. This physiological clearance process is important when dealing with various sugars (clearance is how they are washed out of the mouth). It is often referred to as oral or saliva clearance and the rate of clearance can be measured.

The total volume of saliva is, however, rather small, about 0.8–1.2 ml. Therefore, the film of saliva covering the surfaces of the oral cavity is only about 100 μ in thickness, being thinnest along the hard palate. The saliva film is constantly moving towards the throat to be swallowed. The slowest movement takes place in the labial sulcus of the upper jaw, while the floor of the mouth has a much greater flow (it is also here that the dental nurse aspirates saliva when the patient is in the dental chair). The main stream of saliva runs on each side of the tongue. This has important implications in the clinic because there is limited exchange between these two streams. If applying a fluoride agent on one side of the mouth, the fluoride ion concentration will mainly rise in that part of the oral cavity, and very little is distributed to the other side. We shall return to the possible implications in a later chapter.

The clearance rate varies greatly between individuals, but when chewing, particularly a sweet substance, the salivary secretion rate is enhanced. This is why children chewing sugar cane do not experience a significant increase in caries development. In Figure 2.30 you see Stephan curves showing the pH of saliva during sugar cane chewing, which demonstrates the importance of the salivary secretion and subsequent oral clearance. Both the increased buffer content of the saliva and elimination rate is enhanced when chewing, so the expected drop in pH does not occur despite the mouth being filled with juicy sugars.

You should appreciate that the concentration of sucrose in the thin salivary film will rapidly diffuse into the often much thicker biofilm layer. The diffusion rate depending on the concentration gradient. This gradient is high during the first minutes after exposure and the sugar concentration in the biofilm will therefore remain high despite the oral clearance rapidly diminishing the concentration of sugar in the saliva film. Therefore, elimination through a water rinse after a 'sugar meal' does not eliminate the fermentable sugars from the biofilm interior. It is a useless way to attempt to control caries. Mechanical toothbrushing, which disturbs the biofilm is important after such meals.

In addition to flow rate, buffering capacity, and oral clearance, the importance of the inorganic composition of saliva, in particular the concentrations of calcium and phosphate in resting and stimulated saliva, has already been eluded to. The importance of the degree of saturation and supersaturation with respect to the minerals in teeth cannot be underestimated, and the very dynamics of caries dissolution rates in relation to pH fluctuations must be kept in mind.

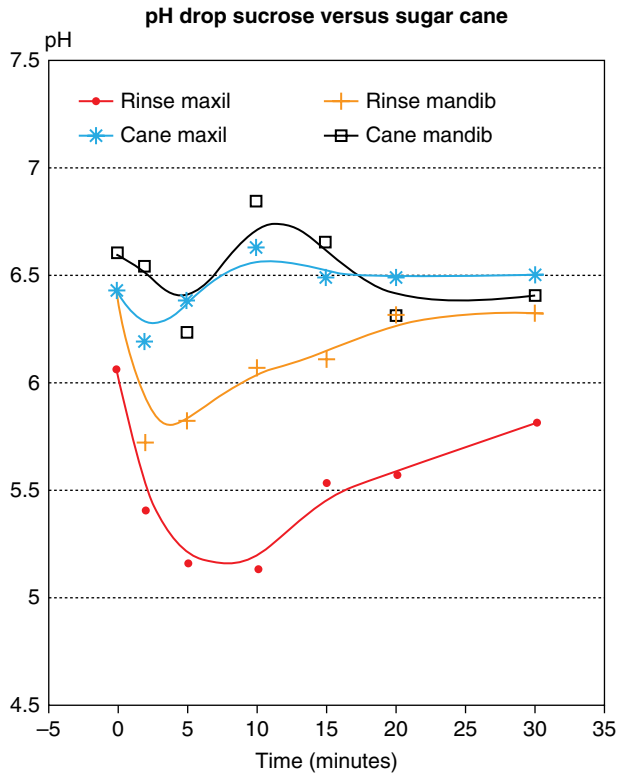

Figure 2.30 Stephan pH response curves following sugar cane chewing accompanied by extensive salivary flow compared with the effect of just rinsing with a 2% sucrose solution and no chewing.

If all this was not bad enough, shortage of saliva also affects the microbial composition of the biofilm. The cariogenic organisms tend to overgrow. Small wonder it is difficult to control caries under all these adverse conditions.

2.7 The role of sugars in dental caries

When the metabolism of the biofilm was described, it was explained that the microorganisms, in order to survive, metabolize sugars and, as a waste product, produce acid. It is this acid that can demineralize the dental hard tissues and this explains why sugar is so important in dental caries. Sugars are types of soluble carbohydrates that provide energy in the diet. Compared with other types of carbohydrates, they are quickly absorbed into the body and are less filling. Sugars are used to enhance the flavours of food and drink, and can be added by

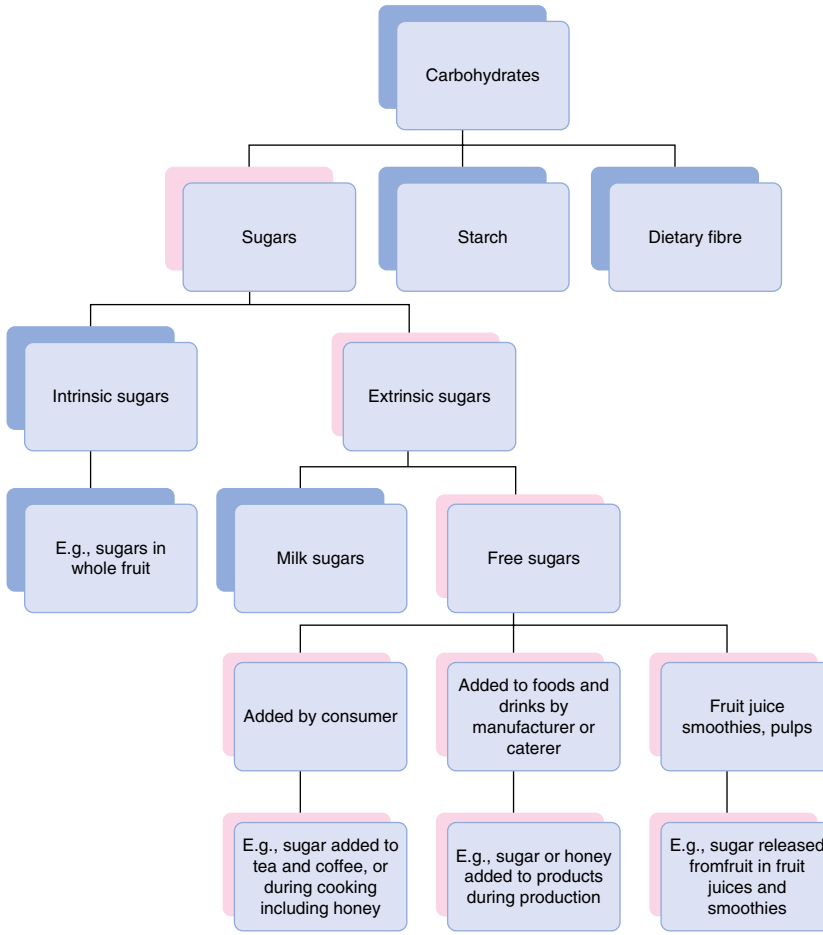

Figure 2.31 Schematic presentation of classification of sugars.

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the consumer or by the food and drink industry. The latter is sometimes known as 'hidden sugar' as the consumer may not know that the sugar is there.

Sugars can be described as either intrinsic or extrinsic (Figure 2.31). Intrinsic sugars occur naturally within the cellular structure of a food and sugars in whole fruit are an example of this. Extrinsic sugars, on the other hand, include milk sugars (lactose) and so-called 'free sugars'. It is these free sugars that can be added to food and drink by the consumer or by the food industry. While intrinsic sugars are not thought to have an adverse effect on general health or dental health, free sugars can be detrimental, and contribute to both dental caries and the obesity epidemic.

So where are free sugars found? To give examples, they are in sugared soft drinks, sweets, cakes, biscuits, sugared breakfast cereals, jams, honey, and preserves. These sugars can be added to, or hidden in, other foods and drinks by the manufacturer, making the product more palatable. Examples of products with added or hidden sugar include baked beans, ready-made sauces, yoghurts, some bread, and soups. The consumer may not expect these products to have sugar added by the manufacturer. Start to look at labels on foods and you will be amazed how many have added sugar. Free sugars are also found in products that state they have 'no added sugars' such as fruit juice, fruit concentrate, smoothies, and dried fruit. Sugars have not been added, but they have been released from the fibrous fruit in the drinks by the juicing process. Patients may well consider these products are healthy and safe for teeth, but they are not and this will be examined in Chapter 4.

'Sugar is the arch criminal of dental caries' is a common statement. There is no doubt that modern diet and its sugar content combined with the excessive use of sugary snacks, soft drinks, and sweet fruit juices play a key role in the development of dental caries, which is ubiquitous in all populations. Therefore, the dental profession in many countries has campaigned against sugars and the widespread use of sugars in so many products to be consumed (much of this sugar being 'hidden', which means that the consumer is not aware of the sugar content in a given product). However, these campaigns have not been very successful. In Denmark, which had one of the highest prevalence and severity of dental caries in the world in the 1950s and 1960s, massive campaigns were run to reduce sugar consumption. In schools, it was possible to try to encourage parents and children to eat sweets and snacks on only one day of the week. Despite this effort, the total consumption of sugar per capita has remained high (around 50 kg/individual/year). Nevertheless, Denmark experienced a more than 90% caries reduction since the middle of the previous century because enormous resources were put into campaigns on oral hygiene and the use of fluoridated toothpastes through the extensive public school dental service. We will explain in a later chapter how this has been possible through caries control. However, just think of how many resources might have been spared, and may be spared in future, if sugar consumption had been restricted. In Chapter 4 there is surprising information why this may not have happened.

At the beginning of this chapter it was explained how dental caries develops and that the addition of sugar to the dental biofilm results in acid formation. The more sugar, and the more frequently sugar is eaten, the more rapidly caries lesions develop. This was convincingly demonstrated when dental students were told to refrain from oral hygiene and concomitantly started to rinse their

mouths nine times a day for 2 minutes with a 2% sucrose solution. (Ethics committees would not allow dental students to be used in such experiments now.) Within a few weeks most students developed early white spot active caries lesions and the experiment had to be stopped. After professional tooth cleaning and topical fluoride treatment, oral hygiene was re-established using a fluoride-containing toothpaste. Most lesions regressed because they were in a very active stage with highly porous surfaces and were partly polished away.

However, it was apparent that the students did not develop lesions at the same rate! Remember there is a great individual variation in saliva composition, rate of biofilm formation, rate of salivary secretion, and so on. It was not because some students had 'stronger teeth' than others. No such thing as enamel resistance to acid attacks exists under clinical conditions, as was once thought. One student in fact developed such extensive demineralization within 3 weeks (Figure 2.21) that it was not possible to bring all of the surfaces back to normal (Figure 2.22). It will now hopefully be appreciated that it is necessary to make sure that in any examination when encountering new **active** caries lesions should be accompanied by a thorough examination of the patient's diet and habits. Chapter 7 explains exactly how to do this using diet analysis as a tool.

With this in mind, it is important to say that it is not only modern life styles that are associated with dental caries. Dental caries is a result of the metabolism in the dental biofilms and these have always existed, throughout history. Dental caries has been found in skulls from ancient people – and even attempts to drill a hole in a tooth with a stone bur, have been found in a skull from an individual living 5000 years BC. In Imperial Rome dental caries was common (Figure 2.32) and there are several reports from medieval populations in Europe about caries experience. Because the food was much coarser and contained small fragments of milled stone from grinding the flour, much of the caries lesion was abraded away occlusally and approximally (it is very similar to contemporary

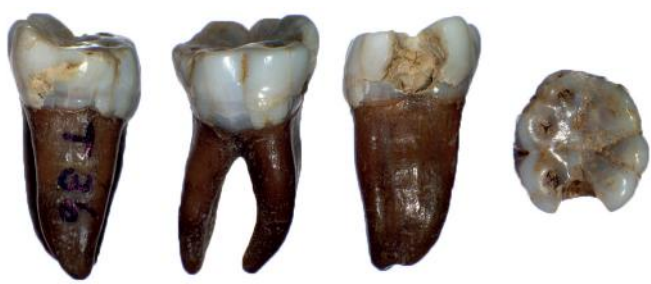

Figure 2.32 A tooth from Imperial Rome 2000 years ago with a large approximal caries cavity.

populations living outdoors in low income countries to day). However, in such populations the abrasion could only keep pace with the slowly progressing lesions due to attrition and when gingiva gradually retracted, approximal spaces were created with large stagnation areas and, here, root surface caries developed 'undisturbed'. These observations from historic material emphasize that dental caries has always been part of human life. However, **contemporary soft diet and industrialized food, with the excessive spread of sugar and fat, are responsible for the dental misery in the last century** because natural attrition of tooth surfaces cannot keep pace with the rate of sugar-induced demineralization. This is why caries control measures are needed in all contemporary populations as long as politicians do not regulate the massive 'sugar pollution' of society.

Finally, and equally important, sugar is partly responsible for the obesity epidemic in many parts of the world. A lack of exercise and a fast food culture is also important and obesity undermines the general health of large parts of the populations. Dental patients can only benefit from this rising concern about sugar intake and dental professionals can play a key role, together with all other health care personnel, in changing this situation. As will be apparent, sugar restrictions and advice therefore is a main constituent of community campaigns for improved health (see Chapter 8) in some contemporary populations.

2.7.1 Sugar substitutes

The sweet taste of sugar is attractive, but sugar is damaging to teeth and is implicated in obesity. Sugar substitutes are sweet, but cause less or no damage. These products may be divided into two categories, those with no calorific value (**non-nutritive** or intense sweeteners) and those with a calorific value (**nutritive** or bulk sweeteners). Table 2.1 lists examples of sugar substitutes.

The **non-nutritive sweeteners** are safe for teeth because they cannot be converted to acid by biofilm microorganisms. They are produced in tablet form

Table 2.1 Sugar substitutes

Non-nutritive	Nutritive
Saccharine	Sorbitol
Acesulfame-K	Xylitol
Aspartame	Mannitol
Thaumatin	Maltitol
	Hydrogenated glucose syrup (Lycasin)
	Isomalt (Palatinit)

and can be added to drinks such as tea and coffee. The drink will not now be cariogenic.

Manufacturers add these products to sugar-free fruit squashes, diet fizzy drinks, and unsweetened sparkling fruit-flavoured water. They are preferable to the sugary alternatives. However, because they are acidic, they contribute to dental erosion. None of these products should be given to infants and young children.

The **nutritive sweeteners** are sugar alcohols. Sorbitol and xylitol are the most commonly used in a variety of sugar-free products, such as sweets, chewing gums, toothpaste, and sugar-free medicines. While sorbitol may be slightly fermented, xylitol is definitely non-acidogenic. Ever since it was introduced in the well-known Finnish studies from the University of Turku, xylitol has been hyped widely and is an interesting alternative sweetening agent.

It has been claimed that xylitol affects bacterial growth and metabolism, de- and remineralization, reduces biofilm formation, and decreases the number of mutans streptococci. Most claims come from laboratory work that has not been confirmed in clinical studies. To truly affect such processes, virtually all other sugars must be replaced in foods and this alone would probably result in constant stomach problems (loose stools) because of its osmotic effect in the gut where it will retain water. It is not fermentable to acid and it is a good sweetener – as is sorbitol – so it is certainly useful in 'no sugar containing products'. Its remineralization potential has been claimed to be supported from chewing gum trials, but appropriate controls have been lacking. Remember it was shown that chewing alone increases salivation, and leads to enhanced carbon dioxide and, hence, buffering in saliva. Thus, claims of a caries-preventive effect for xylitol are so far not substantiated and superiority claims of xylitol over other polyols in the clinic do not seem justified.

Further reading

Bardow, A. and Vissink, A. (2015). Saliva and caries development. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management* (pp. 83–106). Wiley Blackwell, Oxford.

Fejerskov, O. (2015). Pathology of dental caries. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management* (pp. 49–81). Wiley Blackwell, Oxford.

Fejerskov, O. and Larsen, M.J. (2015). Demineralization and remineralization: the key to understanding clinical manifestations of dental caries. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management* (pp. 155–170). Wiley Blackwell, Oxford.

Fejerskov, O., Scheie, A. A., Birkhed, D., and Manji, F. (1992). Effect of sugar cane chewing on plaque pH in rural Kenyan children. *Caries Res*., **26**, 286–289.

Fejerskov, O., Scheie, A. A., and Manji, F. (1992). The effect of sucrose on plaque pH in the primary and permanent dentition of caries-inactive and -active Kenyan children. *J. Dent. Res*., **71**, 25–31.

Marsh, P. D., Takahashi, N., and Nyvad, B. (2015). Biofilms in caries development. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management* (pp. 107–131). Wiley Blackwell, Oxford.

NHS Health Scotland (2012). *Oral Health and Nutrition Guidance for Professionals*. NHS Health Scotland, Edinburgh.

Scheie, A. A. and Fejerskov, O. (1998). Xylitol in caries prevention: what is the evidence for clinical efficacy? *Oral Dis*., **4**, 268–78.

Stephan, R. M. and Miller, B. F. (1943). A quantitative method for evaluating physical and chemical agents which modify production of acids in bacterial plaques on human teeth. *J. Dent. Res*., **22**, 45–51.

Van Loveren, C. and Lingstrom, P. (2015). Diet and dental caries. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management* (pp. 133–54). Wiley Blackwell, Oxford.

Chapter 3

Detection, diagnosis, and recording in the clinic

3.1 What do we need to know and why?

In Chapter 1 it was stressed that while all dental biofilms exhibit intense metabolic activity, only biofilms where a shift in metabolic activity towards an enhanced acid production over longer periods of time, will result in a net loss of mineral from the underlying tooth surface. The reflection or symptom of this is what can be detected with the naked eye and classified as the **caries lesion** on the tooth surface. It was pointed out that lesions may be **active** (if nothing changes in the oral environment, they will progress) or **arrested** (if nothing changes they will stay as they are). Thus, the things it is necessary to know in order to make an appropriate treatment decision are:

- Is a lesion present? This is **detection** of the lesion.
- Is the lesion judged to be active or arrested? This decision, adding the aspect of activity to detection, is **diagnosis**.
- Is the surface of the lesion **intact** or is a **cavity** present? If there is a cavity, can the lesion be cleaned by the patient?

Diagnosis has been called a 'mental resting place on the way to a treatment decision'. For instance, grading a lesion as active implies that the clinician considers that, if nothing is done, the demineralization will progress. Figure 3.1 is a decision tree showing how the diagnostic decision may guide the treatment. Thus, the diagnosis **detects and excludes disease**, assesses **prognosis (considering the entire oral condition of the mouth),** and forms the basis for the **treatment decision**. Lesions where the tooth surface is intact can be managed by the patient's caries control measures. However, a cavity in a tooth may prevent access for the toothbrush. In addition, it may be unsightly and the tooth may be sensitive. These lesions may require restorations as a part of caries control. It is the duty of the professional to **discuss with the patient** whether any action is required in order to control lesion progression. Finally, the diagnosis should allow the clinical course of the disease to be **monitored** at subsequent visits.

This all sounds very simple, but unfortunately it is not quite as easy as it seems. Here, are some of the problems:

- First a trap for the unwary. The lesion is the result of metabolic activity in the biofilm, but paradoxically, this has to be removed in order to see the lesion clearly. Decisions about lesion activity are always made in **conditions of uncertainty**. All diagnostic methods have inherent errors and it is not always possible to separate active from arrested lesions. Various stages in lesion progression reflect a continuum, but it is not always easy to judge where a lesion lies on this continuous scale. The decision is a 'best guess'.
- This is not a disaster because dentists review patients and, at review, the decision is revisited. What, if anything, has changed on review?
- Diagnostic tests need to be **valid**, which implies the test measures what it is intended to measure. For instance, a white spot lesion with an intact surface indicates a lesion that has not yet cavitated.
- Diagnostic tests need to be **reliable**. Reliability or reproducibility means the test can be repeated with the same result. For instance, the dentist would consistently recognize the same white spot lesion with a matt surface as an active lesion. The person should be consistent with himself or herself (intra-examiner reproducibility) and consistent with others (inter-examiner reproducibility).

3.2 Prerequisites for detection and diagnosis

A clinical/visual examination is needed and this requires **good lighting** and **dry, clean teeth**. Plaque should be cleaned off each tooth surface before attempting accurate diagnosis, although it is important not to clean and polish the entire dentition prior to making a diagnosis as a matt surface **underneath** a

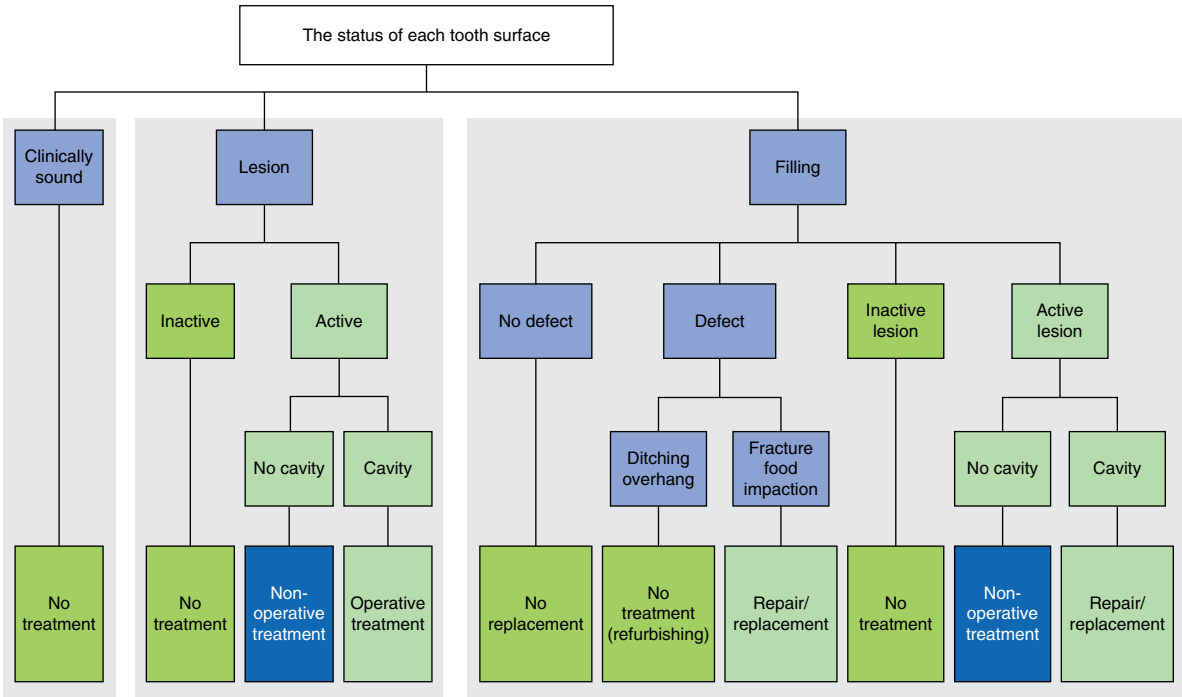

Figure 3.1 Decision-making tree for dental caries including activity assessment as a key factor in the decision process. The flow diagram promotes the concept that active lesions (cavitated and non-cavitated, as well as recurrent lesions) need professional management, whereas inactive lesions do not need treatment besides self-performed toothbrushing with fluoride toothpaste. The flow diagram does not consider individual factors that may influence the modality or intensity of the professional treatment.

Adapted from *Community Dentistry and Oral Epidemiology*, 25, 'Assessing the stage of caries lesion activity on the basis of clinical and microbiological examination', pp. 69–75. Copyright (1997) with permission from John Wiley and Sons. Courtesy of Jim Page.

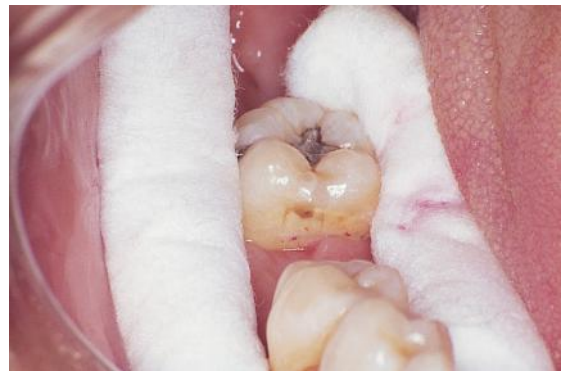

Figure 3.2 Lower right quadrant clean, dry, and isolated with cotton wool rolls prior to clinical examination. Inactive (arrested) caries is present on the mesial aspect of the lower second molar. The lesion probably stopped progressing after extraction of the lower first molar.

substantial dental biofilm adds to the likelihood that the lesion should be classified as being active. Remember to brush the plaque away from occlusal surfaces because it is easy to miss a white spot lesion at the entrance to a fissure unless the surface is clean. This is illustrated in Figure 7.13, p. 160.

Each quadrant of the mouth is **isolated** with cotton wool rolls (Figure 3.2) to prevent saliva wetting the teeth once they have been dried. Thorough **drying** should be carried out with a gentle blast of air from the three-in-one syringe. Alternatively, if not in a conventional surgery, teeth can be dried with cotton wool. White spot lesions are more obvious when teeth are dry (Figure 3.3a,b). **A mirror** should be used to retract the cheeks and lips, and facilitate vision in some areas. Reflected light from the mirror aids vision and may reveal dark shadows, which may suggest dentine lesions (Figure 3.4). Examine every surface of every tooth in the same order, e.g. occlusal, distal, buccal, mesial, lingual. Examine the junction of restorations with the tooth.

Sharp eyes should be used to look for the early signs of demineralization. The side of a probe can be used to remove plaque. Sharp **probes** should never be used

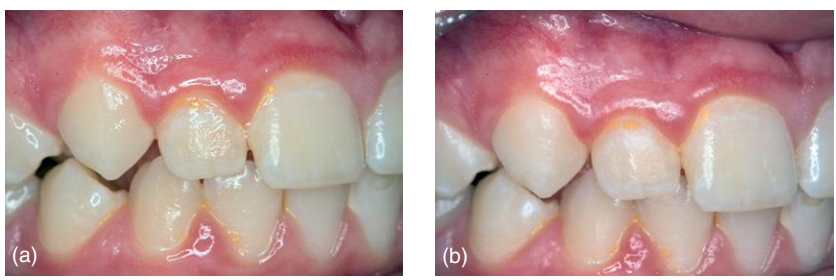

Figure 3.3 White spot lesions on upper central and lateral incisors. (a) Before and (b) after drying, when the lesions become more obvious. The gingivae are inflamed.

By courtesy of N.P. Innes

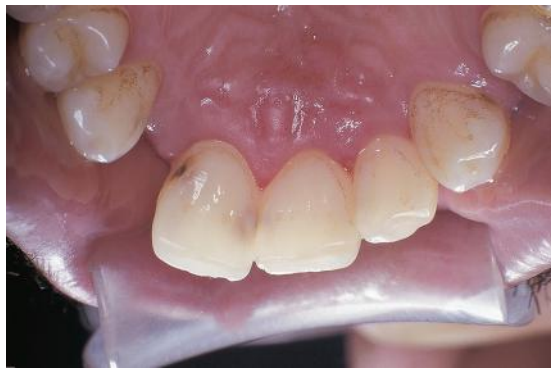

Figure 3.4 Mirror view of the palatal surface of the upper anterior teeth. Lesions are visible as dark shadows mesially in the upper incisors.

to detect the 'tacky' feel of early cavitation, because a probe used with force to prod, can damage a white spot lesion creating a hole, which will subsequently trap plaque (Figure 3.5). However, a sharp probe is useful in diagnosis. It can be used very gently to draw across the lesion and detect the slight roughness of the active white spot lesion. Remember, it is a delicate instrument, not a bayonet! The gentle use of the probe explains why these methods are described as visual-tactile criteria.

For a long time there has been a search for how early it is possible to detect demineralization. This search for the 'truth' is really of no relevance to the health outcome of the patient. With the knowledge about the events taking place that lead to the development of caries lesions, which were introduced in Chapters 1 and 2, you will understand that seeking early loss of calcium and phosphate from the tooth is only of research interest (for example, when seeking an understanding of mechanisms of action of fluorides). Thus, the distinction between 'caries' and 'sound' is indefinable, because it is dependent on the choice of measuring scale.

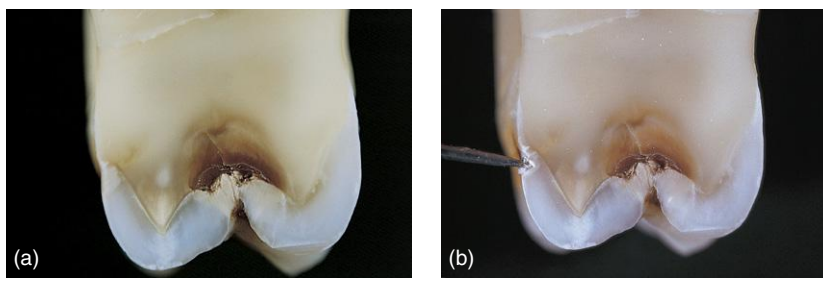

Figure 3.5 A sharp probe has been jammed into the white spot lesion on the buccal aspect of this extracted molar. (a) The lesion before probing. (b) The probe and the resulting damage. On the occlusal surface the enamel lesion has formed on the walls of the fissure. Heavy use of a sharp probe would be similarly destructive on this surface.

Rather, an approach that is sensible and meaningful from a **clinical patient-orientated health outcome perspective** is recommended. There should be a close link between the caries categories that are chosen for detection and the management options available. Only by so doing can the best long-term health outcome for the patient be obtained.

3.3 Commonly used visual criteria

Two commonly applied strategies of clinical-visual diagnosis will be described. One concentrates on lesion activity and integrity, while the other focuses on clinical criteria that predict lesion depth, but do not assess activity. Both have been extensively investigated, used in research studies, and found to be both valid and reliable.

3.3.1 Lesion activity assessment

These are sometimes called the Nyvad criteria after the researcher who first described them. It is a **visual-tactile** (tactile means a probe is involved) method of caries detection designed to allow dentists to monitor lesions over time. The method is based on the surface characteristics of enamel and dentine that change in response to the activity of the overlying biofilm. The surface characteristics addressed are the **activity** as reflected by the surface texture of the lesion and **integrity** as expressed by the presence or absence of a cavity or microcavity.

All lesions, including fillings, are assigned to one of the following diagnostic categories:

1 Active, non-cavitated (Figure 3.6).
2 Active, non-cavitated with microcavity (Figure 3.7).
3 Active, cavitated (Figures 3.8a,b).
4 Inactive, non-cavitated (Figure 3.9).
5 Inactive, non-cavitated with microcavity.
6 Inactive, cavity (Figures 3.10).
7 Filling.
8 Filling with active caries (Figure 3.11).
9 Filling with inactive caries (Figure 3.12).

Active, non-cavitated lesions, are plaque covered, often close to an inflamed gingival margin, and have a matt appearance indicative of surface loss of tissue (Figure 3.6). These lesions may feel rough if the tip of a sharp probe is gently drawn across them. Arrested lesions, on the other hand, may be located free of the gingival margin, as this retracts when not inflamed. These lesions have a plaque-free, shiny, lustrous surface (Figure 3.9). Sometimes they are brown because the porosities have absorbed exogenous stain from the mouth (Figure 3.2).

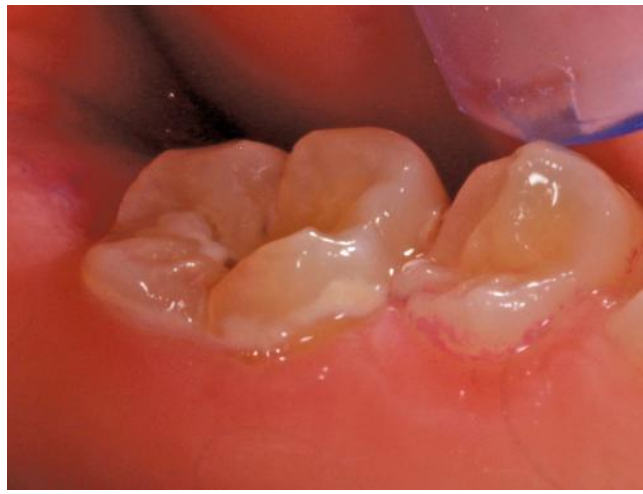

Figure 3.6 Active, non-cavitated, white spot lesion on the on the lingual surface of the lower first molar. The gingivae are inflamed indicating poor plaque control.

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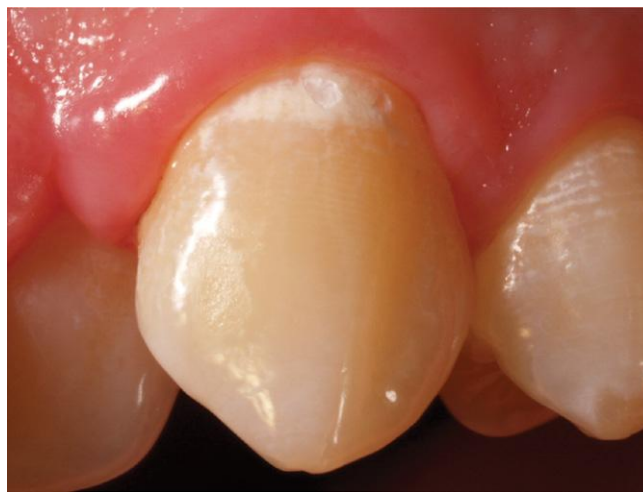

Figure 3.7 Active enamel lesion. The surface is dull and rough. There are two small cavitated areas within the enamel. The lesion is close to an inflamed gingival margin.

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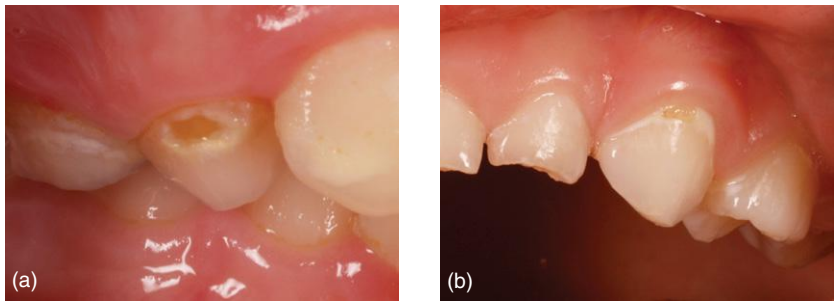

Figure 3.8 Cavitated active lesions on buccal surfaces of deciduous canines. These lesions can be arrested by cleaning alone and fluoride application in toothpaste and/or varnish.

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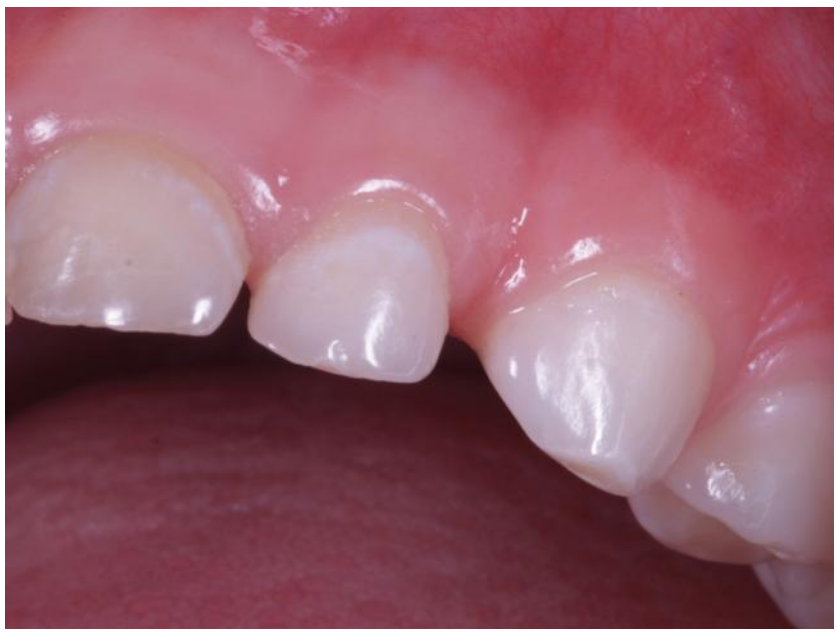

Figure 3.9 Inactive (arrested) white spot lesions. They are shiny, smooth and remote from the gingival margin which is inflamed. Further help with brushing is needed. See also Figure 3.2 where an arrested brown spot lesion is present mesially.

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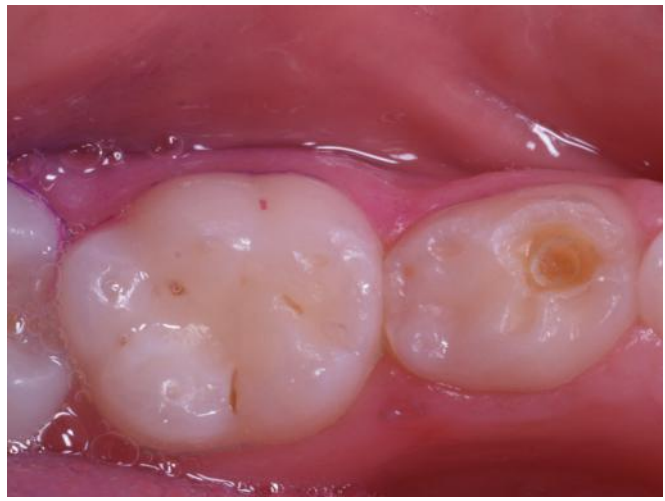

Figure 3.10 Inactive (arrested) occlusal lesion in first deciduous molar. The undermined enamel has fractured away and this lesion is cleansable. Provided the patient and the parent keep this clean, no filling is required.

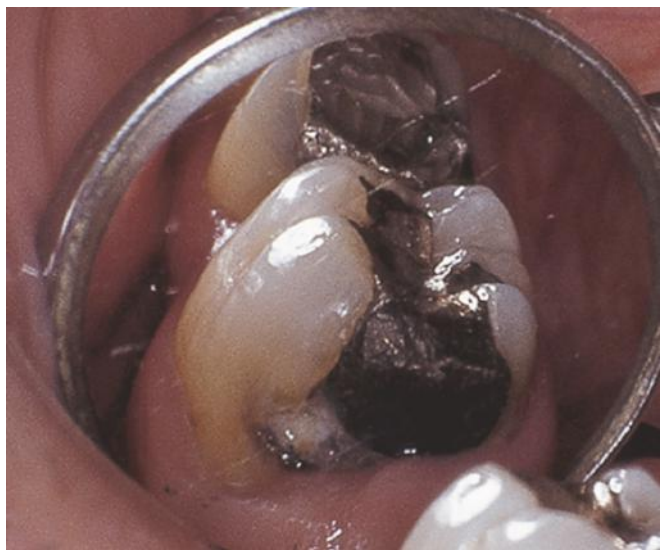

Figure 3.11 Active cavitated lesion, full of plaque, is present at the cervical margin of the restoration in this molar.

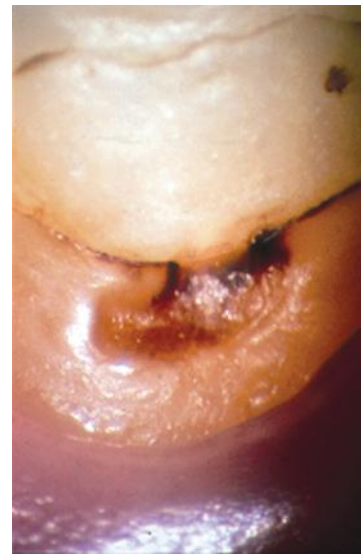

Figure 3.12 Inactive caries lesion at the margin of a tooth coloured restoration. The lesion is cavitated, but hard. It is being controlled by brushing with fluoride-containing toothpaste.

Reproduced from Kidd, E., et al., *Pickard's Manual of Operative Dentistry*, Eighth Edition, 2003, with permission from Oxford University Press

3.3.2 Lesion depth assessment

This system is known as the ICDAS criteria (International Caries Detection and Assessment System). It is a visual ranked scoring system that assesses the depth of lesion penetration, including the non-cavitated stages of caries. The visual changes are related to the increasing histological depth of the lesion. The lesion can be monitored over time. The codes of increasing severity of the lesion in terms of depth are as follows:

0 Sound after air drying for 5 seconds.
1 Opacity or discolouration is hardly visible on a wet surface but distinctly visible after air drying for 5 seconds (Figure 3.13a,b).
2 Opacity or discolouration is distinctly visible without drying (Figure 3.13c).
3 Localized enamel breakdown in opaque or discoloured enamel (Figure 3.13d).
4 Underlying dark shadow from dentine (Figure 3.13e).
5 Cavitation in opaque or discoloured enamel exposing dentine (Figure 3.13f).
6 Extensive distinct cavity with visible dentine (Figure 3.13g).

Figure 3.13a–g shows some of these appearances on occlusal surfaces.

Sometimes it is convenient for the clinician to collapse and simplify these categories as follows:

- Sound (code 0).
- Initial stage caries (codes 1 and 2).
- Moderate stage caries (codes 3 and 4).
- Extensive stage caries (codes 5 and 6).

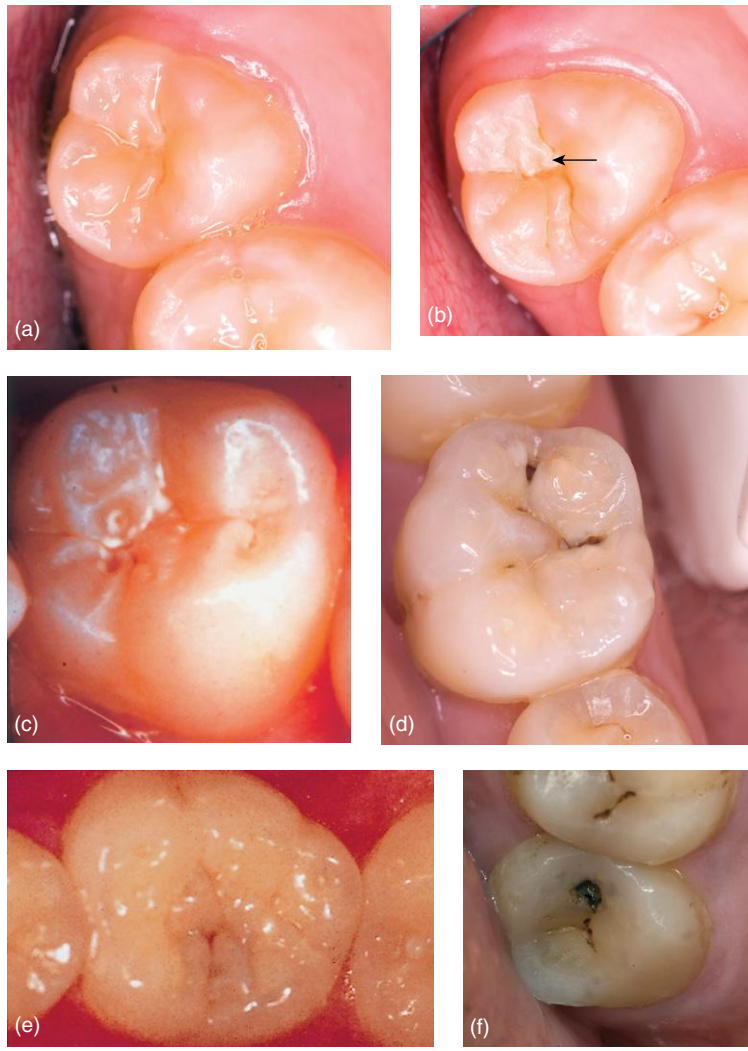

Figure 3.13 (a) Saliva-coated occlusal surface. (b) After air drying, white spot lesion visible (arrow) (code 1). (c) This lesion, on an erupting occlusal surface would be visible on a wet surface (code 2). (d) Localized enamel breakdown (code 3). (e) Underlying dark shadow from dentine (code 4). (f) Cavitation exposing dentine (code 5). (g) Extensive distinct cavity with visible dentine (code 6).

(e) Reproduced from Kidd, E., et al., *Pickard's Manual of Operative Dentistry*, Eighth Edition, 2003, with permission from Oxford University Press.

When using this system each surface must also be given a code for restoration status. These codes are:

0 Unrestored and unsealed.
1 Partial sealant—a sealant that does not cover all pits and fissures of the tooth surface.
2 Full sealant.
3 Tooth-coloured restoration.
4 Amalgam restoration.
5 Stainless steel crown.
6 Porcelain, gold, or preformed metal crown or veneer.
7 Lost or broken restoration.
8 Temporary restoration.

Thus, when recording ICDAS criteria, each tooth surface is designated by two digits. The first digit describes the tooth and the second the caries lesion. For example, an occlusal surface with no restoration or sealant, but a visible white spot lesion on a wet surface would be 02.

3.3.3 Root surface caries

The diagnosis should denote lesion activity and cavitation. Thus, lesions may be:

- Active (non-cavitated or cavitated).
- Inactive (non-cavitated or cavitated).

Active lesions are soft (Figure 3.14) or leathery (Figure 3.15), and are usually found at plaque retention sites next to the gingival margin or along the cement–enamel

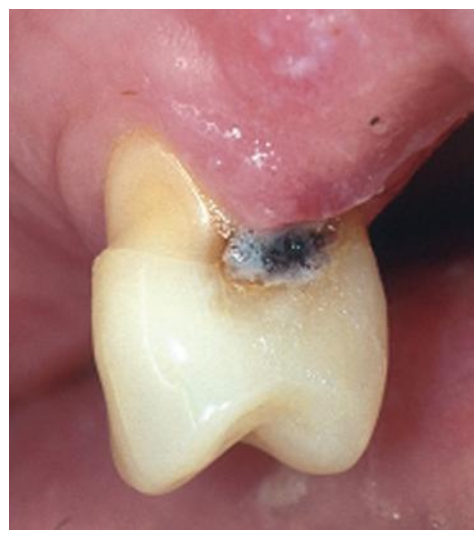

Figure 3.14 An active root caries lesion on the mesial aspect of a premolar. Notice there is no caries lesion on the buccal surface of the tooth. Indeed, this has been so well brushed, it has been partly worn away. The lesion has formed in an area of plaque stagnation next to a removable partial denture. Plaque can be seen in the cavity. This lesion can be arrested by improved cleaning with a fluoride-containing toothpaste and fluoride varnish application.

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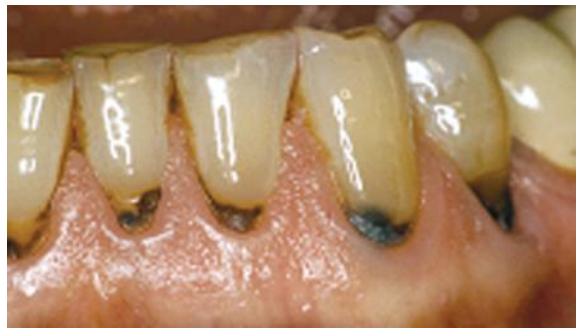

Figure 3.15 These lesions are on the root surface close to the gingival margin. They are darkly coloured and leathery in texture. These are slowly progressing lesions and some are cavitated. This woman is in her 70s. She has a dry mouth and rheumatoid arthritis (secondary Sjögren's syndrome). It is not easy for her to clean, but with help with cleaning, fluoride toothpaste, and fluoride varnish application, these lesions can be arrested.

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junction. Inactive lesions are usually some distance from the gingival margin and feel hard on gentle probing (Figure 3.16). They are often shiny. Colour is not helpful in distinguishing between active and inactive lesions, although typical inactive or arrested lesions tend to appear almost black (Figure 3.17).

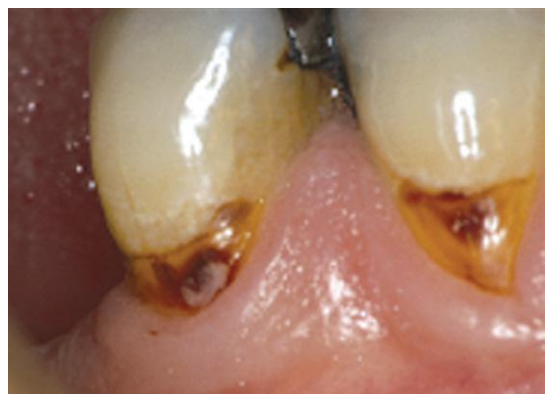

Figure 3.16 An arrested lesion is present on the canine. It is hard and shiny, but not cavitated. A lesion is also present on the first premolar and part of this is cavitated and active. This area is soft and covered with plaque. The remainder of the lesion is arrested. Toothbrushing alone will arrest the active part of the lesion.

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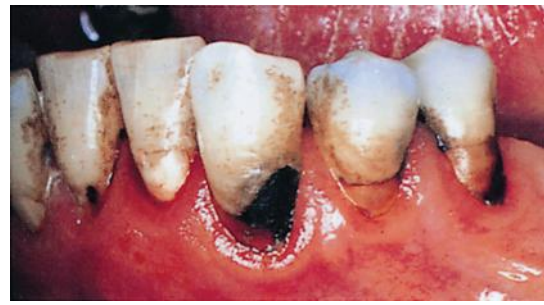

Figure 3.17 Inactive, black root surface lesions.

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3.3.4 Recurrent caries

Recurrent caries is caries at the margins of restorations and these lesion may also be active or inactive (Figures 3.11 and 3.12). The lesions are most often at the gingival margins of restorations (approximal and smooth surface) because this is where the biofilm is most difficult to disturb and may stagnate. Margins of occlusal restorations are easier to clean so that recurrent caries is not common on these surfaces.

A particular problem with amalgam restorations is marginal breakdown or fracture, often called **ditching** (Figure 3.18). This used to be regarded with suspicion by clinicians and restorations were replaced as a preventive measure to avoid plaque stagnation in this area. There are a number of reasons why this approach is incorrect:

- Ditching occurs occlusally in an area that is easy to clean. Recurrent caries usually occurs approximally and cervically where the biofilm stagnates.
- Clinical study has shown ditching does not reliably predict infected dentine beneath the ditched area, unless there is an obvious cavity that would admit the tip of a periodontal probe (over 0.4 mm).

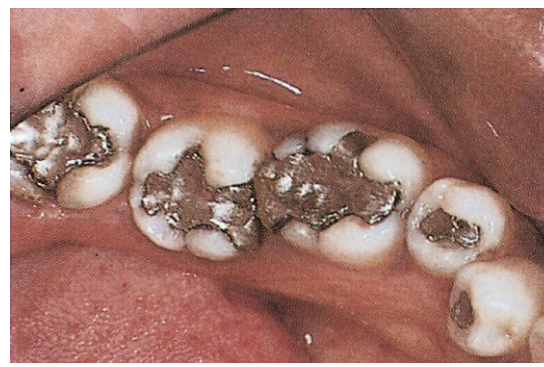

Figure 3.18 Ditched amalgam restorations.

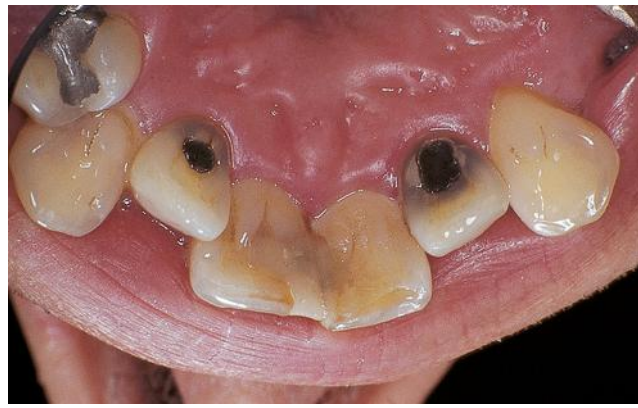

Figure 3.19 The enamel around the amalgam restorations on the palatal aspect of the upper lateral incisors is discoloured. Is this discolouration due to caries or corrosion of the amalgam? A decision was made to replace these restorations and removal of the amalgam revealed discoloured, hard, dentine. This was corrosion and the replacement was unnecessary.

- When dentists remove ditched fillings, they overcut cavities by as much as 0.6 mm. The dentist may also perpetuate the error of cavity preparation, which caused the ditching problem. This is often by creating too sharp an amalgam margin angle, which makes the edge of the filling prone to fracture. The tooth is thus in danger of entering a repetitive restorative cycle until the dentist literally runs out of tooth.

Discolouration around restorations with clinically intact margins also does not reliably predict new caries beneath the restoration. Sometimes discolouration around an amalgam can be caused by corrosion products from the amalgam (Figure 3.19) or by light reflecting from the amalgam itself through the relatively translucent enamel (Figure 3.20). Discolouration around an amalgam may also indicate demineralized, stained dentine, but this is **residual caries** left by the dentist who placed the filling (Figure 3.21). If these restorations are removed, the dentine is discoloured, but either crumbly and dry, and not heavily infected. This does not indicate new disease. Staining around an amalgam restoration should not trigger its replacement or refurbishment, unless there is an active carious cavity or a very wide ditch that is impossible for the patient to clean.

Colour changes around tooth-coloured filling materials may come in a number of forms. An active white spot may be present and caries control measures are indicted. A line of stain at the junction of the filling and the tooth may

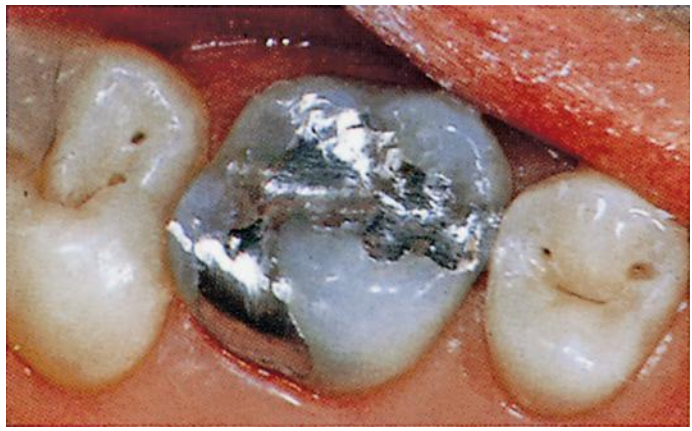

Figure 3.20 New amalgam restoration. Note how the amalgam discolours the tooth. In a few years' time it may be difficult for another dentist to judge whether this discolouration represents the amalgam, corrosion products, or caries. Compare with Figure 3.18.

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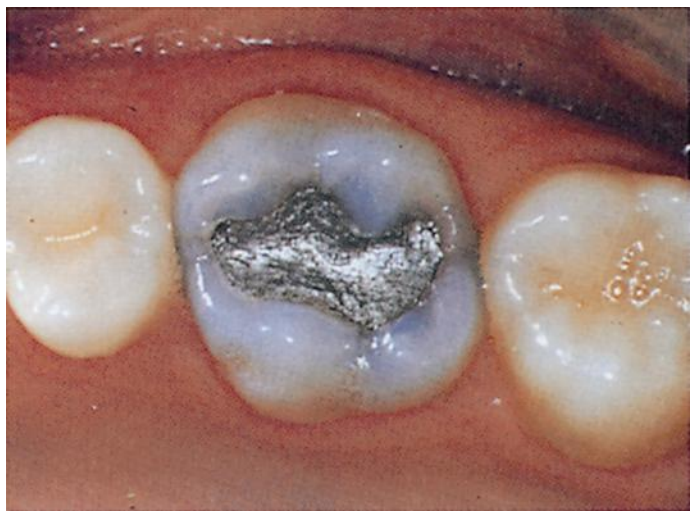

Figure 3.21 The enamel around the amalgam in the upper first molar is discoloured. The restoration was removed to reveal demineralized, but crumbly and dry dentine. This is arrested residual caries (the dentist left it when originally restoring the tooth). If this were recurrent (new) caries, a new lesion would be present next to the filling.

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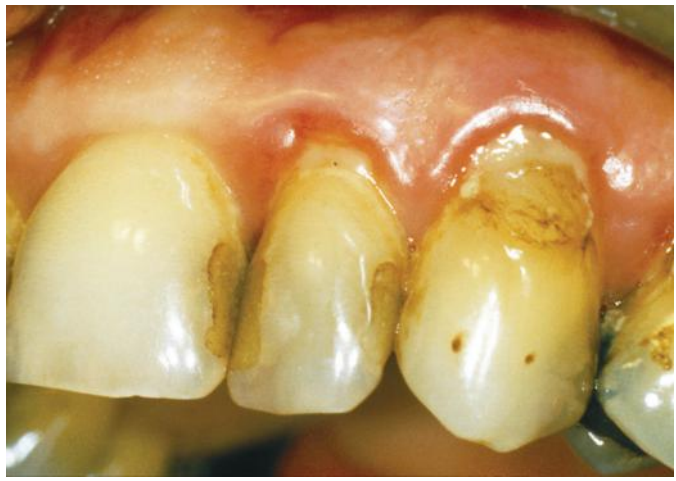

Figure 3.22 Stain around the margins of composite restorations in the upper anterior teeth. This appearance may indicate leakage of fluid around the restoration or caries lesions developing at the filling margins. However, there is no evidence that this reactivates lesions in dentine beneath the restorations. These fillings may be replaced because appearance is a problem. Notice plaque stagnation and gingival inflammation. Oral hygiene instruction will be very important in this case.

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indicate leakage around the filling, but unless the patient requests replacement because of poor appearance, operative treatment is not required (Figure 3.22).

Stain around a tooth-coloured filling can also present as grey or brown discoloured dentine shining through intact enamel (Figure 3.23a). This appearance probably represents residual caries left when the cavity was originally repaired (Figure 3.23b). If the margin of the filling is clinically intact, the filling does not need to be replaced.

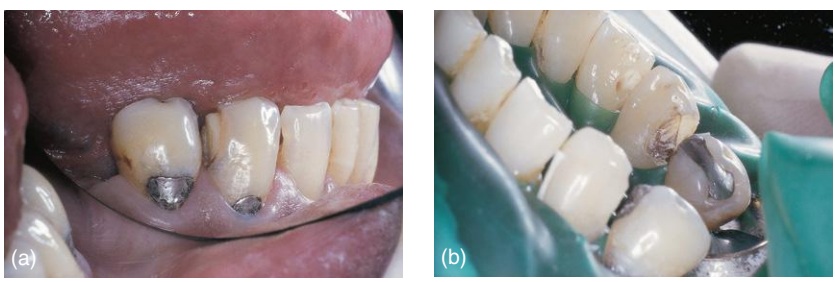

Figure 3.23 (a) Stained dentine around a tooth-coloured restoration. (b) The appearance of the cavity once the restoration has been removed. Stained and demineralized dentine can be seen. If this is hard, or soft, dry, and crumbly, it is likely to be residual demineralization left when the restoration was originally placed.

3.4 Additional aids to diagnosis

It can be difficult to see the lesion on an approximal surface because it forms just cervical to the contact area and vision may be obscured by the adjacent tooth. At a relatively late stage, when it has already progressed into dentine, it may be seen as a pinkish-grey area shining up through the marginal ridge (Figure 3.24). Again, it must be emphasized that teeth must be isolated, clean, and dry to see this.

A sharp, curved probe can be used gently to try to determine if an approximal lesion is cavitated, but take care—if used with a heavy hand this instrument can cause a cavity.

In contrast, an approximal lesion on a root surface may be diagnosed visually, but gingival health is mandatory for such a diagnosis to be reliable. Thus, if gingivae are red, swollen, and tend to bleed, caries diagnosis in these areas should be deferred until improved oral hygiene has been instituted and the inflammation is reduced.

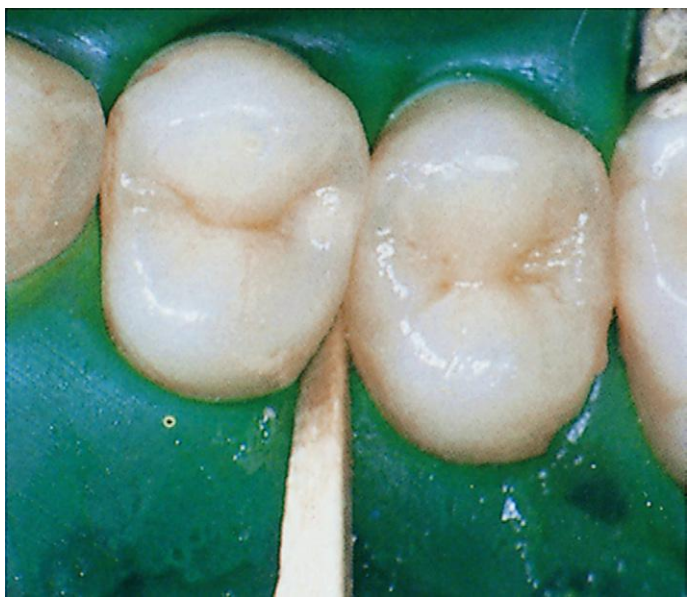

Figure 3.24 There is a distal lesion on the upper first premolar shining up through the marginal ridge as a pinkish-grey discolouration. The lesion is clearly visible in dentine on bitewing radiograph and is to be restored. For this reason a rubber dam is in position. This lesion would be easy to miss on clinical examination.

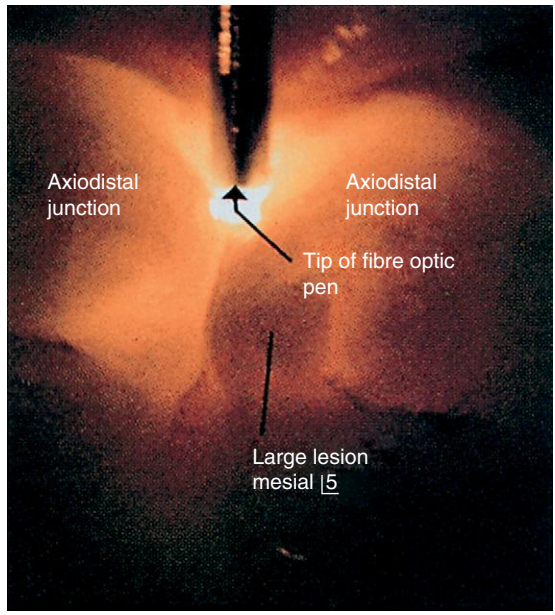

Figure 3.25 Fibre optic light in use to assist in the diagnosis of approximal caries. Shadowing indicates a lesion.

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3.4.1 Transmitted light

This technique consists of shining light through the contact point. A carious lesion has a lower index of light transmission and, therefore, appears as a dark shadow that follows the outline of the decay through dentine. The technique has been used for many years in the diagnosis of approximal lesion in anterior teeth. Light is reflected through the teeth using the dental mirror and carious lesions are readily seen in the mirror (Figure 3.4).

In posterior teeth, a stronger light source is required and fibre optic lights, with the beam reduced to 0.5 mm in diameter, have been used (Figure 3.25). It is important that the diameter of the light source is small, so that glare and loss of surface detail are eliminated. The technique is called fibre optic transillumination (FOTI).

3.4.2 Tooth separation

This technique has been borrowed from the orthodontists who have used it for years to separate teeth before placing bands around them. A small round elastic is forced between the contact points using a special pair of applicating forceps

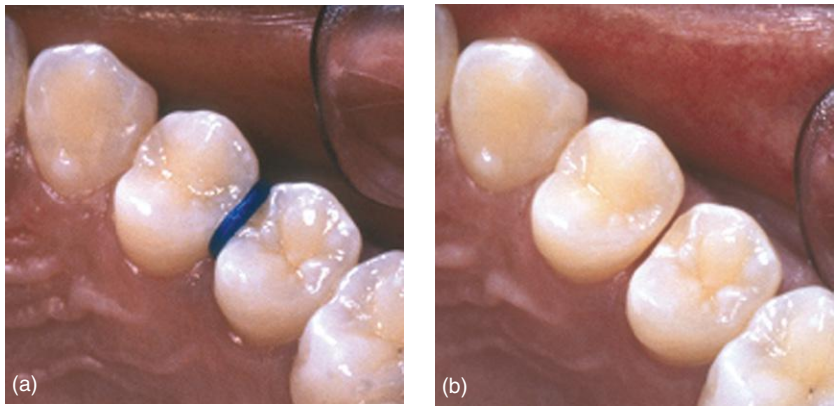


Figure 3.26 (a) An orthodontic elastic separator has been placed between two premolars. (b) After 5 days the separator has been removed and now a probe can be used gently to feel whether a cavity is present.

(b) Reproduced from Kidd, E., et al., *Pickard's Manual of Operative Dentistry*, Eighth Edition, 2003, with permission from Oxford University Press.

(Figure 3.26a). After a few days the teeth are separated (Figure 3.26b). The dentist can now feel, very gently, with a probe to detect whether a cavity is present.

3.4.3 Bitewing radiography

The bitewing radiograph has been used for many years in the detection of the approximal lesion (Figure 3.27a,b). As shown diagrammatically in Figure 3.28, the approximal enamel lesion appears as a dark triangular area in the enamel. The lesion may be in the outer enamel or seen throughout the depth of the enamel. Larger lesions can be seen a radiolucency in the enamel and outer half of the dentine, or a radiolucency in the enamel reaching to the inner half of the dentine. These deep lesions may expose the pulp. Caries on approximal root surfaces is also visible on bitewing radiograph (Figure 3.29).

Occlusal lesions in dentine are also visible on bitewings. The enamel lesion is not seen because the buccal and lingual enamel is thick in this area. However, an obvious radiolucent area in the dentine indicates carious demineralization (Figure 3.30). These occlusal lesions, missed on clinical examination but found on radiograph used to be called 'hidden caries' and the importance of the radiograph used to be stressed. However, lesions of this size **should not be missed clinically provided fissures have been brushed clean and the teeth fully dried**. However, when a bitewing radiograph has been taken to aid the detection of approximal caries, it should always be carefully examined for obvious lesions on occlusal surfaces. Where an occlusal lesion is found on radiograph,

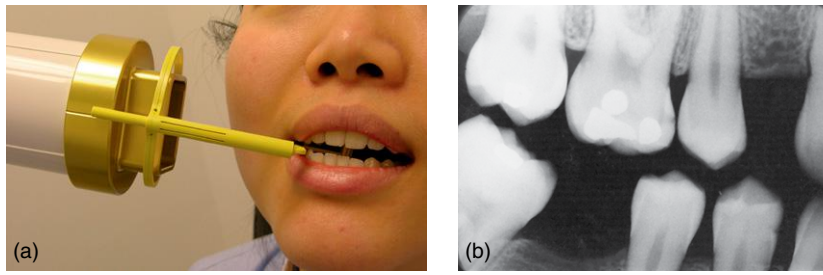


Figure 3.27 (a) A bitewing radiograph is being taken. The film is held lingually by a film holder and the patient closes together on a part of this holder. A beam-aiming device (the yellow arm) helps the operator position the tube so that the beam is directed at right angles to the film. (b) A bitewing radiograph showing caries in enamel and dentine on the mesial aspect of the first molar. A lesion is also visible on the mesial aspect of the lower first premolar.

(a) Reproduced from Banerjee, A. and Watson, T.F., *Pickard's Manual of Operative Dentistry*, Ninth Edition, 2011, with permission from Oxford University Press.

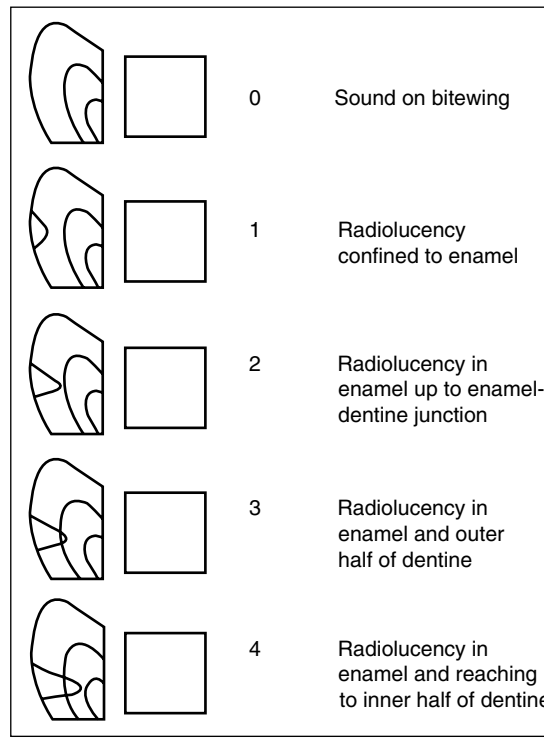


Figure 3.28 Diagrammatic representations of approximal caries on bitewings.

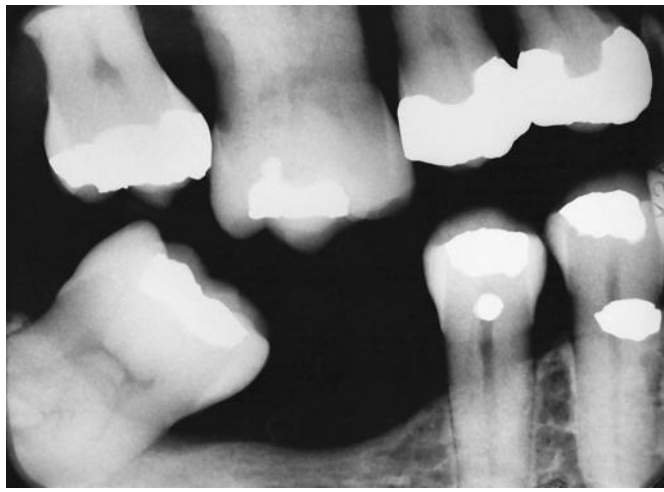


Figure 3.29 Bitewing radiograph showing root caries on the distal aspect of the first upper molar. This tooth has over-erupted following loss of the lower first molar. The patient has been keeping the area clean for years and the lesion is arrested. This is a clinical not a radiographic diagnosis. Remember the radiograph cannot diagnose activity. The lesion should be felt gently with a sharp probe and if it is hard, it is arrested.

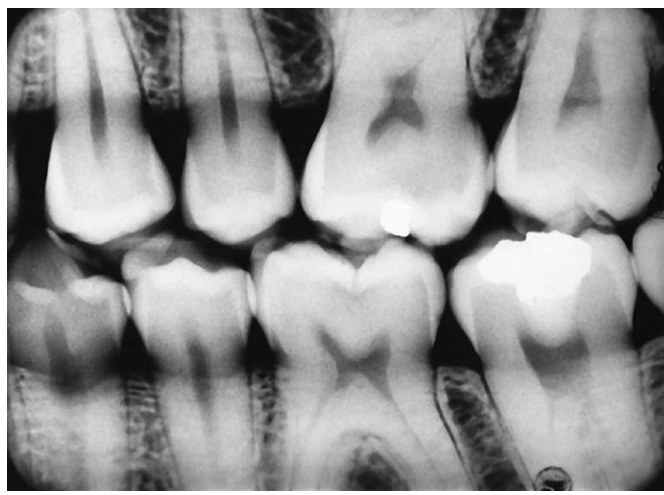


Figure 3.30 Bitewing radiograph showing occlusal caries in dentine in the lower first molar.

go back to check the clinical examination— the lesion may have been missed clinically or the radiograph may have provided a false positive diagnosis.

In summary, the bitewing radiograph aids detection of approximal demineralization, and is advisable in the examination of a new patient with intact contact points. It is an aid to detection, but should never be the only diagnostic tool on which to make a treatment decision. Moreover, it is important to mention that some recent studies in contemporary populations have shown that radiographs only perform better than clinical examination at the cavity/dentine level of diagnosis (Figure 3.31). Perhaps radiographs are not as necessary as once thought!

Certainly the technique has some conspicuous disadvantages in diagnosis:

- Ionizing radiation is involved with its attendant dangers.
- In some situations there will be no facility to take radiographs.

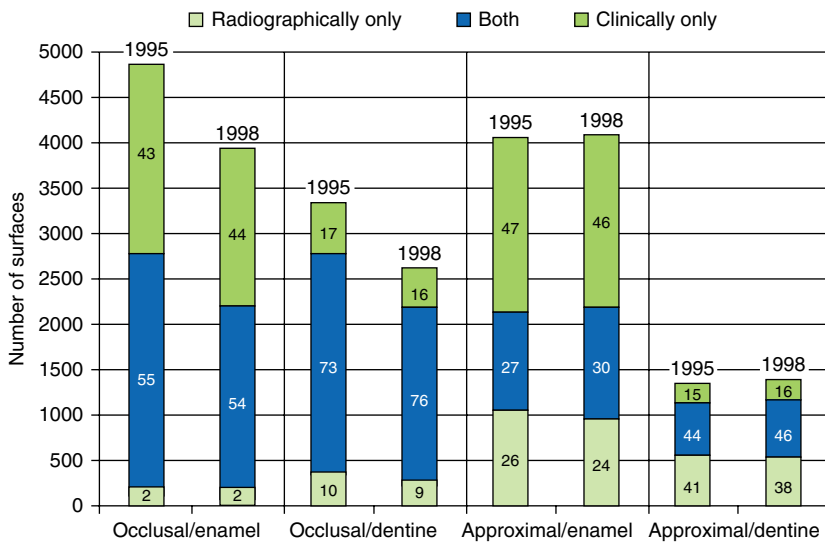


Figure 3.31 Relative diagnostic yields of clinical and radiographic examinations of approximal and occlusal surfaces at the cavitated and non-cavitated levels, respectively. The data were obtained from children examined at 12 and 15 years of age. Note that at the non-cavitated/enamel level of diagnosis, the clinical examination (only), revealed a higher number of lesions than did the radiographic method (only). Only for approximal lesions at the cavity/dentine level of diagnosis did the radiographic method (only) perform better than the clinical examination (only). Age of the individuals did not influence the results.

- While the technique can detect a certain amount of demineralization, it can make no judgement about activity. A series of radiographs, taken over time (perhaps yearly intervals but recall intervals will be discussed again in Chapter 8) are required to confirm the arrest or progression of lesions. It is essential that these views are geometrically comparable and the only reliable way to achieve this is to use film holders and beam aiming devices (Figure 3.27a).
- The technique cannot judge the presence or absence of a cavity, but this is essential information with regard to the treatment required. Cavitated approximal lesions will not be cleansable and restorations are required to aid plaque control. In Chapter 5, the relationship of the appearance on bitewing to the presence of a cavity and the need for restorative treatment, will be discussed further.
- All diagnostic techniques will have false positive results. This means the dentist judges a lesion is present when, in fact, it is not. Be aware that the more diagnostic techniques are used on the same surfaces, the more likely it is that interpretation of one of the techniques will give a false positive diagnosis.

It will be obvious that to be of value, bitewing radiography must be carried out carefully. Overlapping contact points obscure what the clinician is trying to see and unfortunately, slight differences in angulation of the film or X-ray beam will affect what is seen on the resultant radiograph. Thus, radiographs should be as reproducible as possible, using film holders with beam-aiming devices (Figure 3.27a), and standardizing exposure time and dose. This is particularly important when the dentist is going to monitor lesions on radiograph over time to look for progression or arrest of lesions. In addition, films should be read dry, mounted, and under standardized lighting conditions. A report of radiographic findings should always be in the patient's notes.

A variety of lesion detection tools using fluorescent light sources have been advanced in recent years. So far they only have research interest as they do not enable the dentist to make a final treatment decision, which ensures a long-term better health outcome than the proper visual /tactile examination method.

3.5 Charting the examination

The chart in Figure 3.32 is specifically designed to display the Nyvad criteria visually. These criteria focus on the dentist deciding the perceived activity of the lesion and whether the surface is non-cavitated or cavitated. Lesions judged as active should be charted in red and inactive lesions charted in blue. A non-cavitated lesion is indicated by a line and a cavitated lesion by a circle. The chart will allow the health professional to discuss the caries status with the patient. A chart with red lesions indicates active caries and the need for

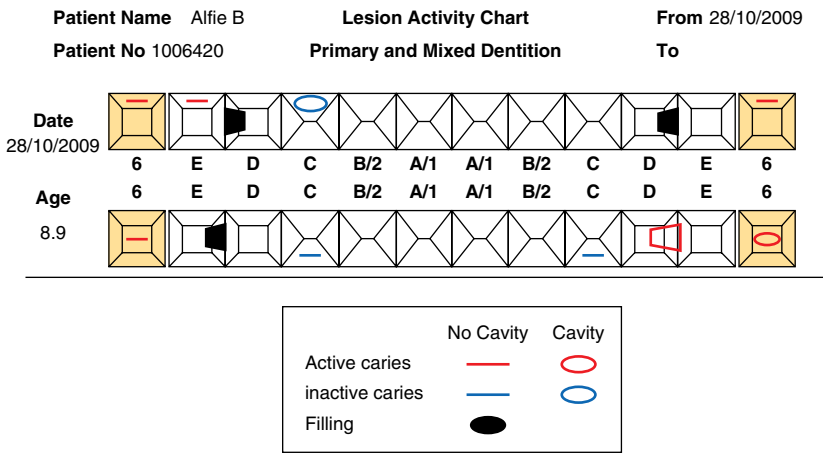


Figure 3.32 Chart with colours to indicate lesion activity (red = active and blue = inactive), and lines and circles (line = non-cavitated, circle = cavitated lesions.) The chart is designed to be updated at subsequent recall visits. This chart was designed, by Jim Page, to record the Nyvad criteria.

increased caries control measures. The chart is designed to be updated on recall and the hope will be that active lesion (red) will be converted to inactive (blue).

The ICDAS criteria, while used in research studies, seem difficult to chart in the clinic in an easily understandable and pragmatic manner. Each surface of each tooth on a chart would be designated with the two digits required (restoration status and caries status).

3.5.1 The authors' preferred diagnostic method

ICDAS criteria may be useful, but the estimation of the depth of the lesion may not be that relevant as the only thing that matters from a clinical decision point of view will be if the pulp is considered vital and able to survive. **We prefer to work on systems that optimize the health outcome by selecting diagnostic methods and categories that result in the best interventions, and thereby the best long-term health outcome for the patient**. We prefer to use the so-called Nyvad criteria (developed by Nyvad and co-workers in clinical practice) because they directly relate to the treatment decision (see Figure 3.1). Active lesions require caries control measures by the patient. Cavities that cannot be cleaned may require restoration (see Chapter 5).

We suggest the following protocol for oral hygiene instruction, caries examination, and charting. The criteria demand the teeth are examined clean and this seems a perfect opportunity to give oral hygiene instruction:

- All patients should bring their toothbrush and toothpaste to the appointment—young children must be accompanied by a relevant carer, preferably the parent.
- Disclose and give oral hygiene instruction. The patient, and if it is a young child, the carer should brush the teeth free from visible plaque. The professional improves brushing if appropriate. At this stage, the professional has the opportunity to check the toothpaste, its fluoride content, how much is put on the brush, how the brush is used, and how the paste is cleared from the mouth. All this can be done by a suitably qualified nurse, hygienist, therapist, or dentist.
- The clean teeth should now be isolated quadrant by quadrant, with cotton wool rolls. The quadrant is dried, and examined for lesions, restorations, and their adequacy. This information is charted.

3.5.2 The relevance of uncertainty to the decision

At the beginning of this chapter it was explained that diagnosis is a 'best guess', carried out in conditions of uncertainty. This is inevitable because lesions present a continuum of activity so the judgement is: 'If nothing changes, do I think this lesion will progress?' However, the condition of uncertainty should affect the decision. If in doubt whether a non-cavitated lesion is active, judge it as active and institute non-operative caries control measures. Nothing is lost by this approach. However, if in doubt about cavitation and whether a filling is needed, give the lesion the benefit of the doubt. Prescribe non-operative caries control measures and review, rather than prescribe a filling, an irrevocable decision.

3.6 Categorizing caries status

Following history, clinical, and radiographic examination, the dentist should categorize the patient into an appropriate caries category. It can be useful to use a coloured tab on the notes to represent this group. This colour coding can be helpful for the dental team caring for the patient and for the patient themselves who must be aware of why their caries status has been categorized in this way.

The suggested groups are:

- Caries controlled (green).
- Caries only partly under control (some active lesions), but all relevant factors can potentially be changed (orange).
- Caries control insufficient—active lesions and some risk factors cannot be changed or appear to be unidentified (red).

In the two latter caries active groups, it is sensible to try to list the factors that seem to be responsible. This will be explored further in Chapter 7. Some factors may be amenable to change, such as oral hygiene and diet. Others may be more difficult to modify, such as medications or diseases that cause a dry mouth.

Further reading

Baelum, V., Nyvad, B., Grondal, H-G., and Fejerskov, O. (2015). The foundations of good diagnostic practice. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management*, pp. 173–190. Wiley Blackwell, Oxford.

Hintz, H., Lussi, A., Cuisinier, F., and Nyvad, B. (2015). Additional caries detection methods. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management*, pp. 211–231. Wiley Blackwell, Oxford.

Nyvad, V. and Kidd, E. A. M. (2015). The principles of caries control for the individual patient. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management*, pp. 303–320. Wiley Blackwell, Oxford.

Nyvad, B., Machiulskiene, V., Soviero, V. M., and Baelum, V. (2015). Visual-tactile caries diagnosis. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management*, pp. 191–210. Wiley Blackwell, Oxford.

Page, J. and Kidd, E. A. M. (2010). Practical suggestions for implementing caries control and recall protocols for children and young adults. *Dental Update*, **37**, 422–432.

Topping, G. V. A. and Pitts, N. (2009). Clinical visual caries detection. In:Pitts, N.B. (ed.) *Detection, Assessment, Diagnosis and Monitoring of Caries*, pp. 15–41. Karger, Basel.

Chapter 4

Control of caries lesion development and progression

4.1 The concept of caries control

The first three chapters of this book have introduced the basics of what dental caries is and how to detect lesions. The next chapter will consider the concept of caries control and begins by explaining why throughout this book the preferred term is **caries control**, rather than prevention. Remember, the formation of the dental biofilm, and its metabolism is an ubiquitous natural process; it cannot be prevented. So:

Question: Who is susceptible to caries lesion development?

Answer: Everyone with teeth, from cradle to grave because the metabolism in the dental biofilm is an ubiquitous, natural process. Lesion development and progression, which may occur over time, are symptoms of the process. We should aim to control these processes so that the development of a clinically visible lesion is avoided. However, if clinical lesions develop and progress these symptoms can be arrested by controlling the environment.

Thus, all patients with teeth should know how lesions may form and progress, and how to control this. Please note the emphasis on the patient. It is the patient who controls caries with the support and encouragement of the professional.

The goals of medicine (and dentistry) are to promote and preserve health if it is impaired, to restore health, and minimize suffering and distress. These goals are embodied in the word 'prevention'. It is agreed that, with dental caries, this is basically what the dental profession is doing—and has always been doing. In many ways this has become a mantra—the dentists rightly claim that they are conducting prevention when recommending the population to eat less sugar, use fluorides, brush teeth, and when lesions occur, drill and fill, in order to restore the dentition and reduce pain and discomfort. Unfortunately, when dentists go for restoration—without ensuring that the patient understands how to control further caries lesion development—they indirectly stimulate the repair cycle, which ultimately may lead to loss of teeth (see Chapter 5). Sometimes the filling may be described as 'treatment' to contrast it with 'prevention'. The dentist is paid for fillings (treatment) and minimally rewarded for so-called prevention. Hopefully, the dentist will have had time to mention that the patient should remember to brush teeth and use fluoride. Do they know how fluoride works? Have they checked whether the patient is able to clean all caries-prone sites? Perhaps the dentist has said 'remember not to eat too much sugar', but how often has the patient been asked to keep a diet sheet? We consider this caries control management to be the **non-operative treatment of caries** and the word treatment is used to imply something that is skilful, time-consuming, and worthy of payment.

This chapter will briefly introduce the caries control managements **appropriate for all patients**. The important factors that everyone can intervene with every day in caries control are:

- Plaque control.
- The use of fluorides.
- A sensible, but not draconian, diet.

4.2 The role of oral hygiene in caries control

Is oral hygiene (plaque or biofilm control) important in caries control? Yes, it is of prime importance. Caries lesions form as a result of the metabolic events in dental plaque. Thus, plaque control is the cornerstone of non-operative treatment. Fluoride-containing toothpaste should be selected with fluoride content never less than 1000 ppm F. Brushing twice daily interferes with the growth and ecology of the biofilm—disturbs biofilm maturation—and fluoride application

retards the rate of lesion progression and in the next section its mode of action will be explained.

The benefits of toothbrushing can be maximized if the following principles are followed:

- Brushing habits should start as soon as the first deciduous tooth erupts. Children need to be helped and supervised by an adult when brushing, even up to 12 years of age.
- Brush twice daily, last thing at night and at one other time each day.
- Children under 3 years should use toothpaste containing no less than 1000 ppm (parts per million) fluoride, the parent putting a small smear of paste onto a small brush. Children should not be allowed to eat or lick toothpaste from the tube. The relevance of preventing small children ingesting fluoride will be explained in the next section under the heading fluorosis.
- From about 3 years onwards, the family fluoride toothpaste (1350–1500 ppm fluoride) is indicated and now a pea-sized helping of paste is used.
- Children should always learn to spit out the toothpaste, and not swallow it, although very young children cannot spit effectively and will swallow most of what is on the brush.
- Rinsing with water after brushing should be discouraged in order to maximize the topical effect of the fluoride; 'spit, don't rinse', is the relevant advice.

4.2.1 Oral hygiene instruction

All patients should have their oral hygiene checked when they visit the dental team. If we are serious about disease control, the patient should attend each clinical appointment with their brush and toothpaste. Everyone—patients and staff—should consider this part of the practice philosophy. Someone in the practice should be checking that the toothpaste contains active fluoride and that children under 3 years have a paste containing 1000 ppm fluoride. Disclosing is very useful. In Figure 4.1a,b the teeth looked plaque-free, but disclosing showed oral hygiene could be better. If the practice philosophy is that oral hygiene is always checked, the patient is likely to attend having done their best, and they should be shown and advised how to improve cleaning in areas where plaque is present.

Should a particular brush or technique, be recommended? If the dentition appears plaque-free and there are limited signs of gingivitis, the individual is either in the hands of caring adults or themselves able to perform an acceptable oral hygiene control. However, if an area is missed by the brush, perhaps the technique in that area needs modifying or perhaps the brush is wrong, perhaps too big. Patient and professional are partners in this effort and the patient is the

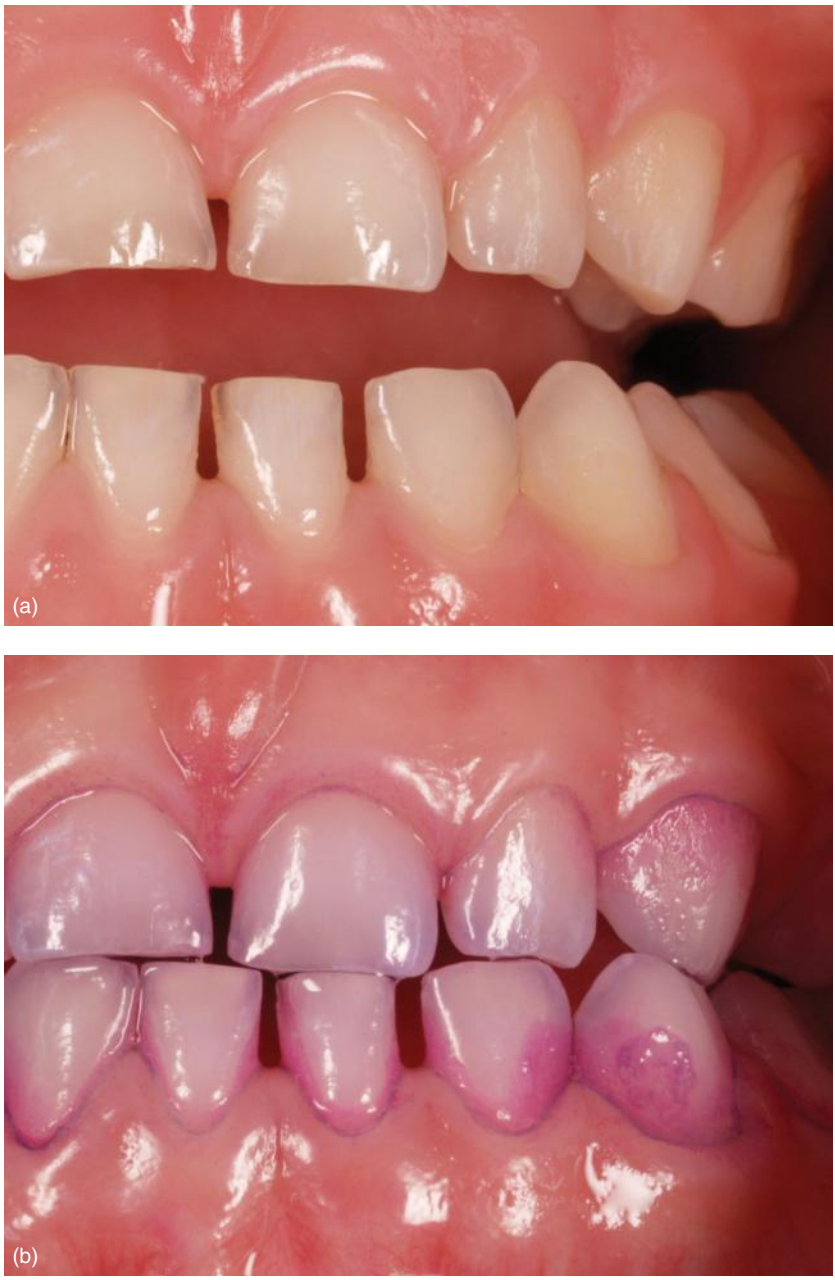


Figure 4.1 Apparently clean teeth (a), but disclosing shows plaque (b). This is the perfect time to give oral hygiene instruction that is relevant and patient specific.

Reproduced from *Dental Update* (ISSN 0305-5000), 37, Page J, et al., 'Practical Suggestions for Implementing Caries Control and Recall Protocols for Children and Young Adults', pp. 422–432. Copyright (2010) with permission of George Warman Publications (UK) Ltd. Courtesy of Jim Page.

Figure 4.2 A disposable mouth mirror allows an adult patient to see plaque on lingual and approximal surfaces.

boss, it is their views that matter. Perhaps the patient would be helped by having a small mirror to see better (Figure 4.2). Ask their opinion.

Toothbrushes

Reviews of evidence have concluded that **powered toothbrushes** with an oscillating/rotating movement (Figure 4.3) are more effective in removing plaque and may improve compliance. Some models have timers built-in, which gives

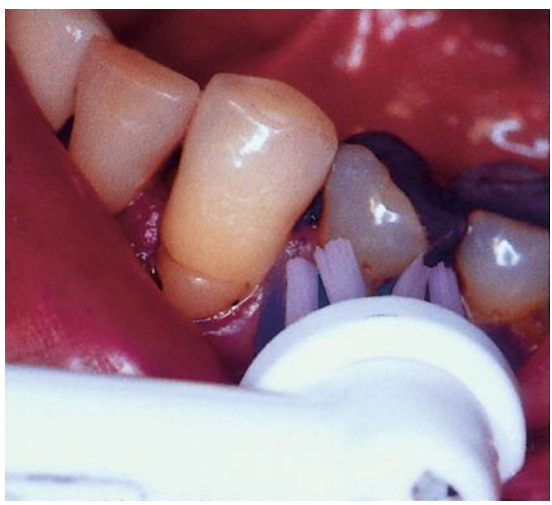


Figure 4.3 A powered toothbrush with a small, circular head which performs rotating movements.

useful feedback to the user on the time spent brushing. Powered toothbrushes can be very useful for patients who are physically handicapped and those with a disability that makes brushing difficult.

Manual toothbrushes vary widely in the shape and size of the head, material, texture, and arrangement of filaments, as well as the size and shape of the handles. Brushes should have:

- A handle appropriate to the age and dexterity of the user.
- A head size appropriate to the user's mouth—a brush with a small head is generally recommended.
- A compact arrangement of medium–hard, rounded nylon filaments.
- Bristle patterns that enhance plaque removal—brushes with bristles arranged at different heights and angles are available.

What matters is that the brush chosen by the patient should be effective for them. Thus, the professional should only suggest a change if they, or more importantly, the patient perceives a problem. Manual brushes and heads of powered toothbrushes should be replaced regularly because, once the bristles are permanently bent, the brush is unlikely to be cleaning effectively.

Brushing methods

When using a powered toothbrush, the brush does the moving and it must just be held in the correct position and moved after an appropriate time.

Using a manual brush it is more difficult, but it does not really matter how a toothbrush is used provided plaque is removed effectively without trauma to the gingival tissues, and without damaging the hard tissues because of the abrasives used by manufacturers. Two particular methods have been described according to the motion performed by the brush:

- A **scrub** method uses a horizontal scrubbing motion and is a useful method to teach children and for use by everyone on occlusal surfaces.
- The **Bass** method where the bristles are directed apically and then placed at an angle to the gingival margin so that when the bristles are flexed the tips are forced between the teeth (Figure 4.4). The brush is then vibrated either in an anterior–posterior direction or by a rotary movement of the handle, keeping the tips of the bristles in position. This method would be advocated in patients with open interdental spaces because it facilitates penetration of the brush filaments. In order to clean the lingual surfaces of the upper and lower anterior teeth it may help to turn the brush vertically.

If the patient has difficulty, it may be useful to suggest one of these methods. When suggesting something new, it may help to put a hand over the patient's hand so that they can feel the motion required.

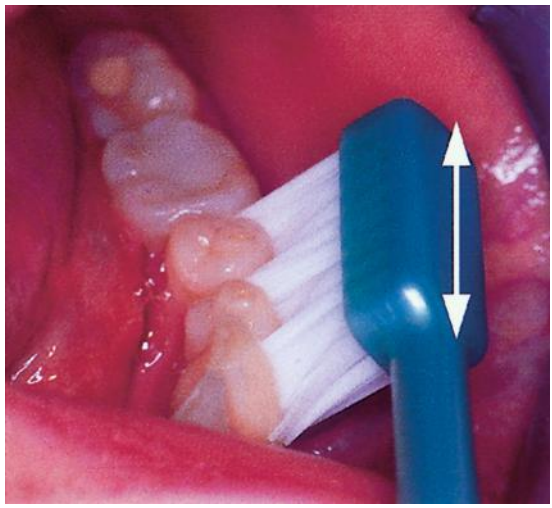


Figure 4.4 The Bass method of toothbrushing. Note the angulation of the bristles against the tooth surface and the direction of the vibratory movement.

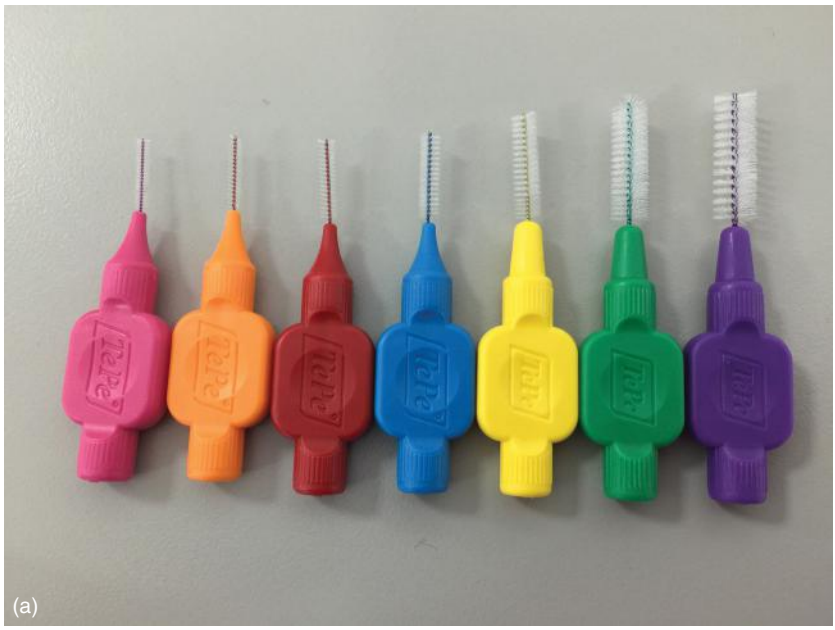
Interdental cleaning

To facilitate plaque removal on approximal spaces the use of dental floss is recommended. Where there has been gingival recession in adults and the elderly with age as a consequence of periodontal disease, there may be wide interdental spaces that the toothbrush cannot access. In these situations, an appropriately-sized interdental brush (such as a Tepe brush) is ideal (Figure 4.5a,b). The dental professional can help select the right size of brush (they are colour-coded), and check the patient is using a mirror so he/she can see and is able to angle the brush correctly.

Toothbrushing for babies and toddlers

As soon as teeth erupt parents/carers should brush them with a smear of paste. A paste with a bland taste, that does not foam excessively, is ideal. One formulated without sodium laurel sulphate is preferred because this detergent causes foam, which the child does not like and, therefore, spits out. Avoid sweet tasting pastes—the development of a sweet taste should not be encouraged.

Supposing the parent tells you the baby will not allow this—they scream, wriggle, or pull the brush away. Toothbrushing is mandatory, but how can you best advise the parent? Well for a start be sympathetic, even if your own child was/is a compliant blob and gives no problem. It should be suggested that the parent should never become angry, as this will hinder, not help. However, the parent needs to understand that not brushing is not an option. With a baby, advise the parent to cuddle the baby in their arms and with a finger gain entry to the closed lips and follow this with the brush. Brush gently explaining



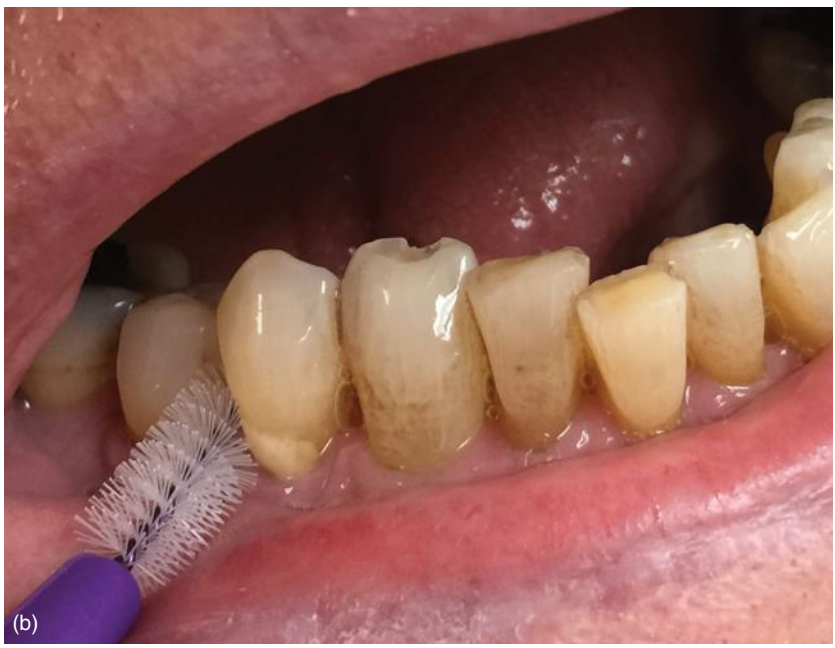


Figure 4.5 Interdental brushes come in many sizes (a). The use of an interdental brush (b).

quietly that brushing is essential just like washing. Show the parent the position of the fraenum over the central incisors and advise avoiding this with the brush as it can be sensitive.

Once the child is 1–2 years old, there are ways of helping this toddler if they are reluctant. The choice will be up to the parent, who will know what may work with their child. Here are some possibilities:

- Link the brushing to a favourite programme on iPad or television—brushing becomes associated with a treat.
- Let the child brush the parent's teeth and then reverse it.
- Buy a toothbrush the child particularly likes.
- Have a sticker chart in the bathroom, with the child choosing and placing the sticker at each brushing.
- As a last resort, use gentle force. Insist, as you would insist on washing a dirty face—be gentle but insist, quiet but firm.

4.3 The role of fluoride in caries control

In the section on plaque control, it was stressed that everyone should be using a toothpaste containing fluoride. However, much more knowledge about fluoride is necessary, including:

- The history of how fluoride came into dentistry.
- How fluoride ingestion during tooth development can cause a defect of enamel structure called dental fluorosis.
- How fluoride influences the caries process—the cariostatic mechanisms of fluoride.
- The clinical efficacy and cost-effectiveness of fluoride.
- Various fluoride vehicles.
- The toxicity of fluoride.

For many years it was thought that, in order to affect dental caries, fluoride must be incorporated into developing enamel. Later, it will be explained that this is not correct and an understanding of this is incredibly important because it dictates how fluoride should be used! An understanding of these mechanisms is needed because it may be necessary to explain them to other Public Health workers and to politicians.

4.3.1 The history of how fluoride came into dentistry

Fluoride has played a key role in the prevention and control of dental caries for over 60 years. It has been used all over the world and in a variety of ways including:

- Fluoridation of water supplies.
- Addition to domestic salt, milk, chewing gum.
- Prescribing it to children as tablets.
- In community programmes, where it has been professionally applied as gels, varnishes, or used in supervised rinsing programmes;
- The addition to toothpastes for regular daily use on an individual basis.

There is no doubt that the discovery of the anti-caries properties of fluorides constitutes one of the most important landmarks in the history of dentistry, but how did it all start? Strangely, perhaps, it started by dentists noticing the unpleasant appearance of enamel formed in some areas in America. The enamel looked mottled (Figure 4.6) and it was later noticed that this unfortunate appearance was associated with less dental caries. The terminology in use today to describe this appearance is **dental fluorosis.** However, when this was first described it was not known that fluoride, occurring naturally in drinking water, was responsible for a disturbance in tooth formation that resulted in the mottled appearance.

In the 1930s experimental animal studies and human epidemiological studies gradually resulted in the establishment of a cause-and-effect relationship between dental fluorosis and fluoride in the drinking water. The epidemiological studies were led by an epidemiologist and dental officer, H. Trendley Dean. In his studies on the relationship between fluoride in water supplies and dental

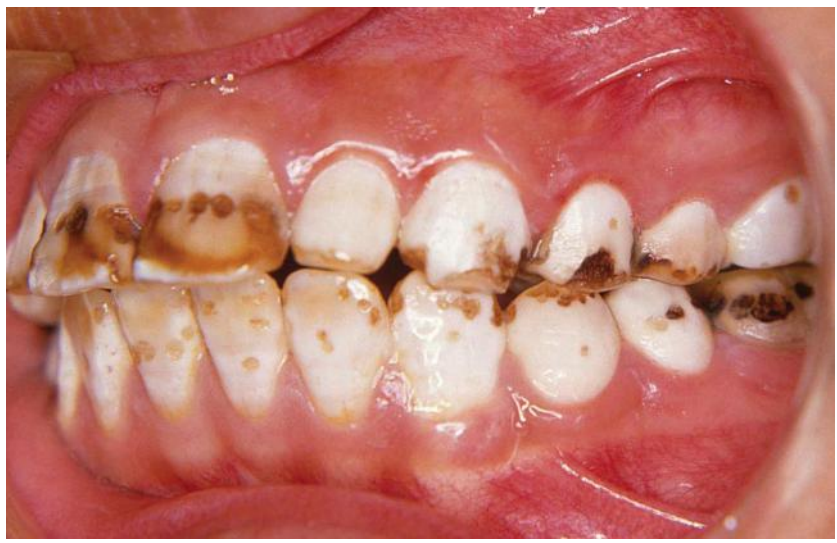


Figure 4.6 Dental fluorosis was originally designated mottled enamel. Note how the different tooth groups are exhibiting variations in degree of clinical severity.

fluorosis, he also noted that a slight increase in water fluoride was associated with a lower prevalence of dental caries. These findings led to considerable excitement about the possibility of adding fluoride artificially to water supplies, in order to lower the caries experience. An attempt was made to add fluoride to water and similar reductions to those observed in naturally-fluoridated water supplies were revealed (Figure 4.7). This sparked the era of artificial water fluoridation in the United States in particular. Today, about half the population in USA lives with artificially-fluoridated water.

Since Dean had already shown that fluoride had a certain toxic effect upon developing dental enamel (dental fluorosis), much thought was given to the question of what might constitute 'the optimal level of fluoride' in drinking water when artificial water fluoridation was to be recommended. Dean attempted to find a level where he could observe maximum caries 'protection', while causing minimal dental fluorosis. He concluded that 'amounts not exceeding one part per million (ppm) expressed in terms of fluorine (F) were of no public health significance'. His work showed that, even below this level, there were mild changes in enamel, but he described these as 'of no public concern' and scored them on clinical examination as 'questionable'.

In any population, some will have concerns about the environmental side effects of adding 'artificial' compounds to drinking water. Anxiety was expressed about the long-term consequences of adding fluoride to water, although in most waters it occurs as a trace element. Some have even alleged fluoride might cause cancer, although such allegations have absolutely no scientific foundation. However, public understanding of the issues involved has probably not

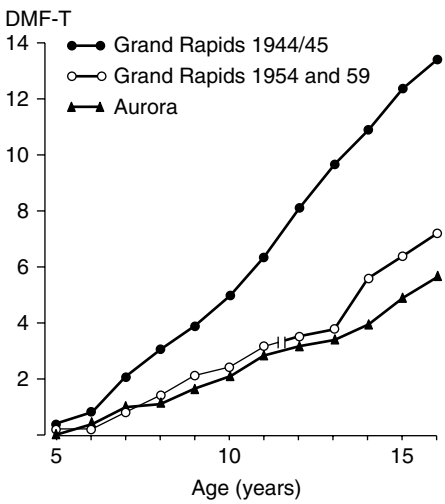


Figure 4.7 Dental caries (expressed as decayed, missing, and filled teeth (DMFT) per child) in Grand Rapids, USA, before water fluoridation (1944–45) compared with Aurora (1945–46), where there was a higher fluoride content in the natural water supply. Ten and 15 years after artificial water fluoridation in Grand Rapids, the caries levels have been reduced by about 50%.

been helped by the dental profession being reluctant to speak openly about the toxicological properties of fluorides. The profession often divided the population into 'pro- and anti-fluoridationists' and has downplayed, or been reluctant to accept, the toxicological effects. Dean's clinical classification of 'questionable' was either claimed not to be caused by fluoride alone or disregarded. Some public health dentists preferred to only talk about dental fluorosis as a cosmetic problem, ignoring the biological effect on mineralizing tissues. All this was done with the best of intentions, but it has led to the public suspecting that the profession has 'something to hide'. As a result the use of fluorides in caries control, in any form, has often been discredited.

Until the 1980s it was believed that fluoride had to be incorporated into developing enamel to exert its maximum effect. Therefore, water was an obvious vehicle because everybody drinks water and developing enamel in children would contain fluoride, and it was claimed they would benefit. When it was not possible to convince the public (for example, in most of Europe, and in Japan and China) fluoride was (and still is) in some countries added to tablets. The dose was designed to mimic what was expected to be ingested when living in areas with 1 ppm F in the water. (**Note that, in contrast to physiological and pharmaceutical principles, we are not truly talking about dosage in terms of mg/kg body weight**.) Similar approaches were taken by adding fluoride to domestic salt and milk.

A breakthrough came when fluorides were added to toothpastes to be sold over the counter as a cosmetic product. Unfortunately, for many years fluoride added to toothpaste was seen as inferior to systemic ingestion because it was believed that to work the fluoride must be incorporated in developing enamel. We now know this is totally incorrect and this will be further explained in section 4.3.2 'Dental fluorosis' when our current understanding of how fluoride works in caries control is considered.

4.3.2 Dental fluorosis

Dental fluorosis is an enamel **hypo**mineralization along the entire enamel surface. The more severe the ingestion has been from birth, the deeper the zone of hypomineralization stretches into the enamel (Figure 4.8). This means that fluorosed enamel is porous and, hence, may take up stain. The biological explanation for this effect is not entirely clear, but it appears that enamel maturation, from the end of enamel secretion until tooth eruption, is very sensitive to fluoride, which affects mineral deposition over this long period (2–4 years).

Clinically, dental fluorosis manifests itself as a pearl-like appearance in the very mild form and, if the teeth are dried, a fine pattern of enhanced striae of Retzius can be observed across the enamel surface (Figure 4.9a). Slightly more

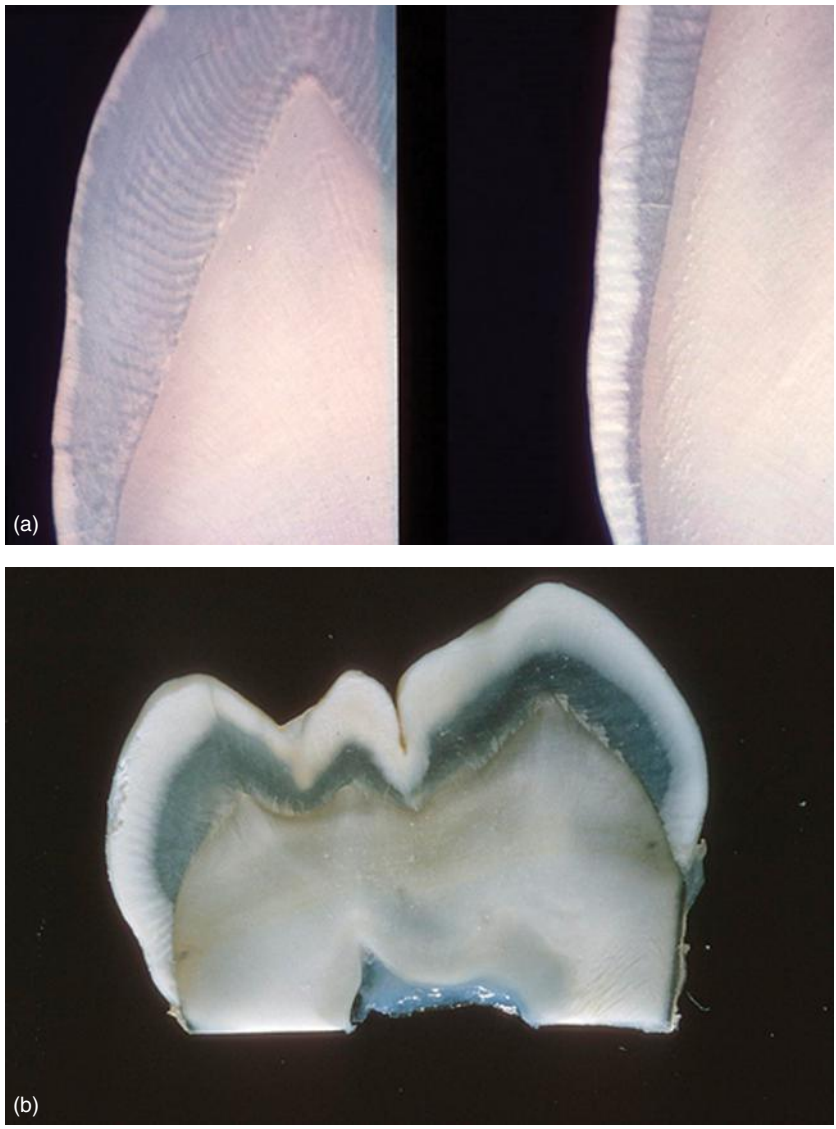


Figure 4.8 Sections of teeth with different degrees of dental fluorosis. Dental fluorosis is a hypomineralization (less mineral content than normal enamel) the severity of which depends on the amount of fluoride ingested during tooth formation. Note: it affects the entire length of the enamel, but clinically it will present differently depending on where it is in the tooth. Cervically, for instance, where the enamel is thin, the hypomineralization extends almost to the enamel dentine border. This appearance contrasts with the much thicker parts of the enamel where the substantial lack of mineral extends only half way through the thickness of the enamel.

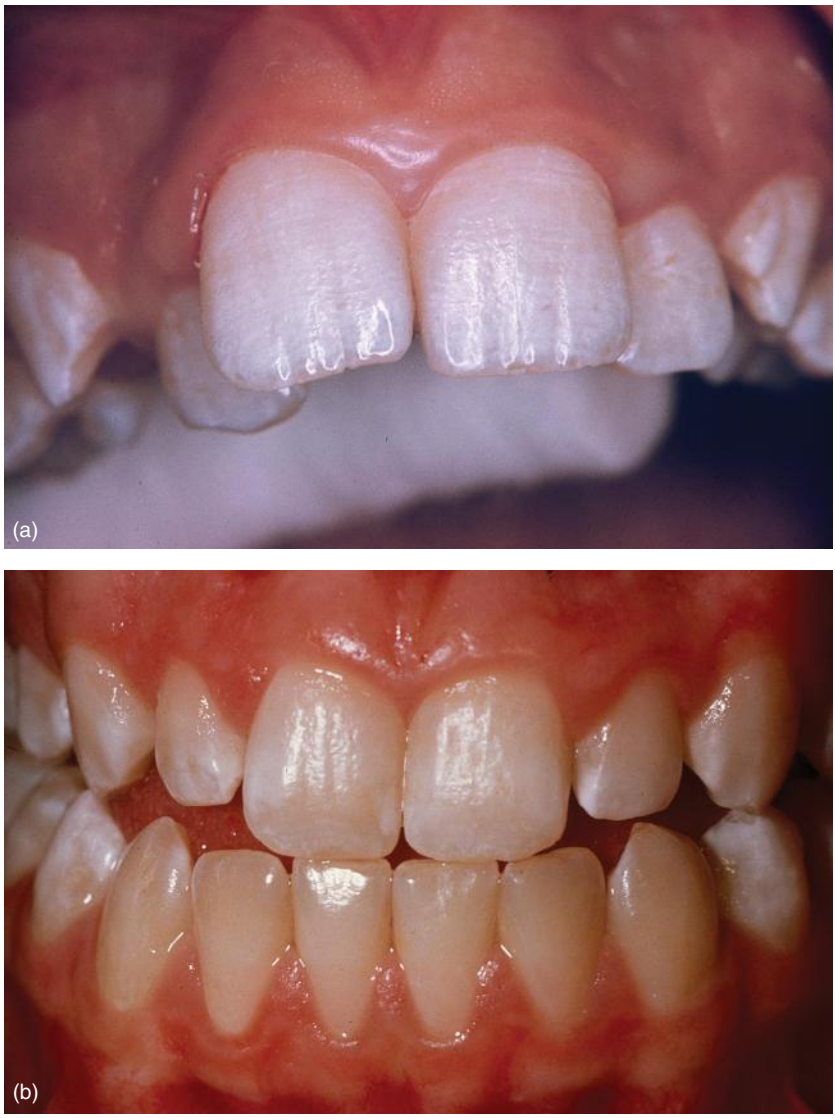


Figure 4.9 Dental fluorosis can be graded by the TF (Thylstrup/Fejerskov) index. The milder forms (TF scores 1–2 compare with Figure 4.11) of dental fluorosis vary depending on the structural variation of normal enamel. The first signs of hypomineralization are seen as fine white lines reflecting the striae of Retzius across the enamel (a). These lines may merge and, in some patients, there are parts of the enamel that are a more homogeneous white. This phenomenon is called snow-capping if found on cusp tips or, as here, in the lateral rim of the upper lateral incisor (b).

fluoride ingested will result in more whitish teeth because these fine lines merge and gradually form larger areas or 'clouds' along the tooth, which are white/opaque (Figure 4.10). These changes can be classified and scored from 1 to 9 as seen in Figure 4.11. This index is called the Thylstrup Fejerskov (TF) index after the dentists who devised it. The examples shown in Figure 4.9a,b represent mild forms and may be scored TF 2 and TF 3.

Teeth develop from before birth until the end of adolescence. Thus, not all teeth are equally affected. The later a tooth is formed and erupts, the more severe dental fluorosis is because fluoride accumulates in the bones. In growing children in particular the bone 'sucks' the fluoride, so it rapidly accumulates in small children and growing youngsters. However, bone constantly undergoes remodelling as children grow up, i.e. small parts are resorbed and new bone is formed, and thereby an equilibrium of fluoride between blood plasma concentration and bone is achieved. Thus, if living with a constant fluoride exposure in the environment, individuals will accumulate fluoride lifelong and

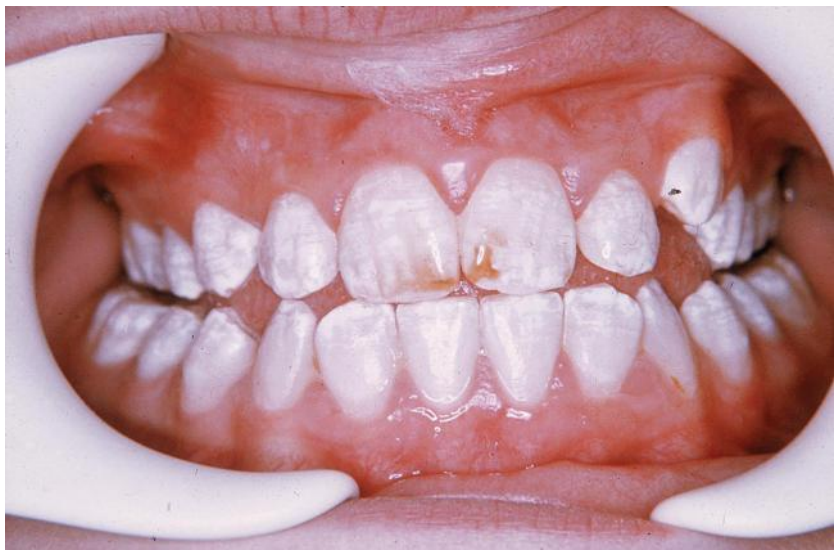


Figure 4.10 In TF scores 3 and 4 (compare Figure 4.11) the enamel surface appears whiter, and because of the increased pore volume (hypomineralization) of the fluorotic enamel these porous, white areas may take up brown stain from foods. When the degree of hypomineralization is more severe, the enamel is so fragile that the mechanical burden of chewing results in fractures of surface enamel shortly after tooth eruption. The porous areas thus exposed, which were subsurface before, now appear dark due to uptake of stain as seen in Figure 4.6.

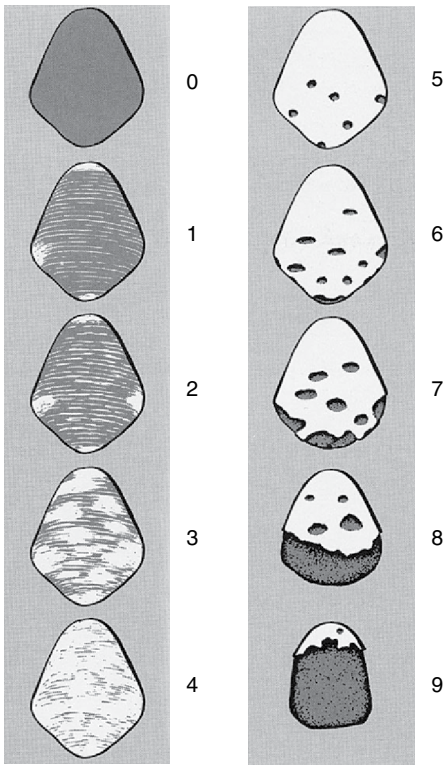


Figure 4.11 Diagrammatic illustration of the TF index. This ranks the clinical features of dental fluorosis from its mildest form—TF score 1—to the most severe—TF score 9. It is the most valid and precise way of recording dental fluorosis.

the plasma concentration will steadily increase. Therefore, in the permanent dentition, anterior teeth and first molars show the mildest degrees in any individual (most fluoride ingested is trapped by the rapidly growing skeleton). Both dentitions may be affected, but fluorosis is rarely, if ever, seen in the primary dentition as a result of fluoride use in caries prevention programmes. The notable exceptions are salt fluoridation as used in South America and after fluoride tablets use (see 4.3.5, 'Fluoride vehicles').

Twenty years ago the actual dosage of waterborne fluoride in the different age groups and in different climates was estimated. These data were compared with the original data from three major epidemiological studies on dental fluorosis with known fluoride content in the drinking water (US studies from the 1940s, 1960s, and 1980s). The results are presented in Figure 4.12. The figure plots the community index of fluorosis (Dean's way of averaging his scores—something that later has been shown to be statistically erroneous) against the daily dose of fluoride (mg/kg body weight).

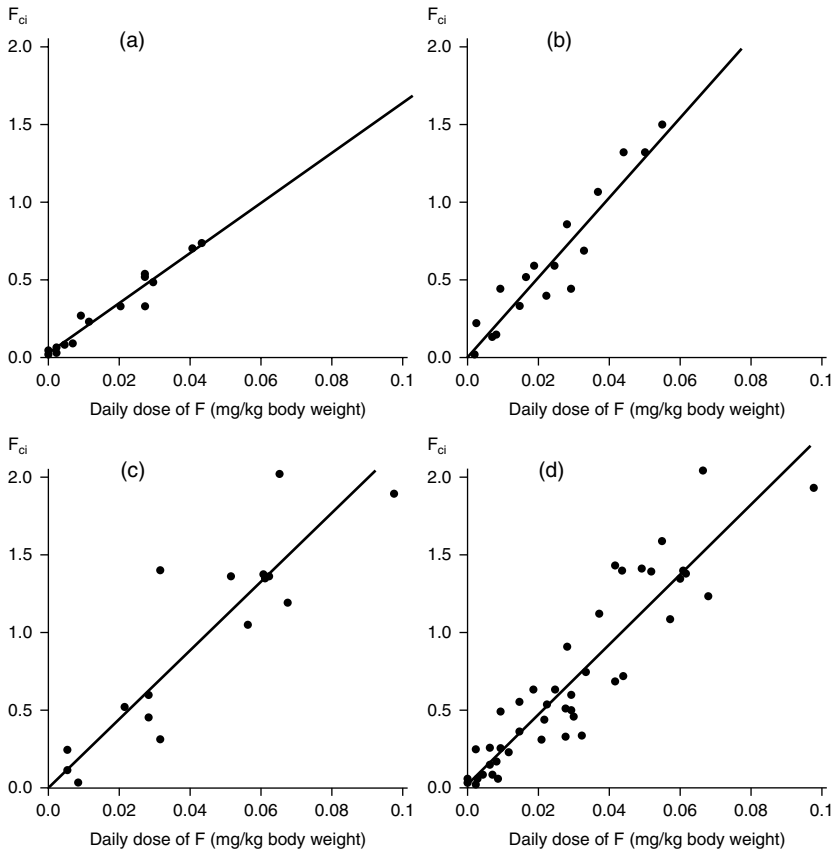


Figure 4.12 Relationship between the daily dose of fluoride ingested in mg/kg body weight and the resulting severity of dental fluorosis as calculated using the index devised by Trendley Dean and called Dean's Community Index (FCI). The data originate from 3 clinical studies conducted in the US in (a) 1941/4, (b) 1967, and (c) 1985. (d) All data sets are pooled together.

Data from *Journal of Dental Research*, 69, 1990, Fejerskov, O. et al., 'The nature and mechanisms of dental fluorosis in man', pp. 692–700.

Please note three features of these graphs:

- Even with very low levels of fluoride intake, a certain level of dental fluorosis will be found.
- The dose–response relationship is clearly linear.
- The more fluoride ingested from birth, the more dental fluorosis that may be expected on reaching adulthood and developing a permanent dentition (assuming access to the same water sources).

Similar calculations have been possible from different countries using fluoride tablets. The same dose–response associations have been shown. Thus, it is possible to estimate the expected level of dental fluorosis in a population 6–12 years ahead if a new fluoride regime is introduced that involves systemic ingestion of the fluoride. However, these calculations have never been done when, for example, introducing salt fluoridation or new tablet programmes!

4.3.3 Cariostatic mechanisms of fluorides

Prior to 1980 it was believed that fluoride exerted its cariostatic effect by being incorporated into the enamel during tooth formation. This meant that fluoride had to be ingested from such sources as water, tablets, or salt. For an optimal caries reducing effect, the fluoride should be consumed from shortly after birth. This mechanism was a logical assumption because chemical data had shown that fluorapatite was less soluble than hydroxyapatite and fluoride accumulates in the outer surface enamel during development.

However, over the years, inconvenient inconsistencies appeared in this argument. For example:

- When a child moved from a low fluoride area to a fluoridated water area, he/she achieved the same caries rate (new lesions developed per time unit) as children who had been born and reared in the fluoridated area.
- Water fluoridation was as effective in adults and the elderly moving into areas with water fluoridation as it was in children reared in the area.
- There was no association between the fluoride concentration in enamel (assessed by biopsies) and caries experience.
- Painting of sound enamel surfaces with 2% sodium fluoride (NaF) solutions had a cariostatic effect without fluoride being incorporated into the sound enamel apatite.
- Could a small difference in surface fluoride concentration between teeth from individuals born in an area with 0.2 and 1.2 ppm F really chemically explain a 50% caries reduction (as was seen in the middle of the previous century)?
- There is more fluoride incorporated into enamel in the surface of a caries lesion than in surrounding sound enamel.
- With age, fluoride accumulates in cervical enamel (which is covered by dental biofilm) even if there are no clinical signs of caries lesion development.

These observations and chemical laboratory experiments during the 1970s resulted in a different explanation.

Fluoride exerts its clinical caries preventive effect by being present in the oral fluids during the dynamic inorganic chemical processes taking place during pH fluctuations at the interface between the biofilm and tooth surface.

In other words, the incorporation of fluoride into the enamel apatite was not the 'cause', but a result of these numerous ongoing pH fluctuations, which gradually may result in mineral loss and lesion development (Figure 2.11).

Suddenly fluoride was seen as an agent that could be used with equal success **in all age groups** and **applied in many different ways**. Very importantly, fluoride programmes with topical applications could be introduced without the constant dilemma of balancing the risk of developing dental fluorosis with obtaining maximum benefit in form of caries reduction.

Fluoride was suddenly not a magic solution that 'prevented' caries, but **an agent that could be used selectively in caries control because it was documented that if fluoride is available in slightly increased concentrations in oral fluids, it will interfere with the chemical events at the surface of the tooth, so that the rate of caries lesion development is slowed down**. Fluoride alone is not the solution, but a very important adjunct in the caries control!

4.3.4 The clinical efficacy and cost-effectiveness of different ways of using fluoride

This section begins by defining what is meant by the terms 'efficacy', 'effectiveness', and 'cost-effectiveness':

- **Efficacy** is the extent to which a specific intervention, method, or procedure produces a beneficial effect under **ideal** conditions. The determination of efficacy is based on the results obtained from randomized controlled clinical trials (RCCT).
- **Effectiveness** is if the methods chosen do what they are intended to do in **real life** in a defined population.
- **Cost-effectiveness** adds the dimension of the manpower, money, and time expended to achieve the results.

Comprehensive reviews (often compiling every paper published uncritically) on fluoride use in caries prevention and control have been published in the last decade. Much of the clinical research conducted over the last half century has been of lower methodological quality than has previously been acknowledged. This statement reflects the fact that epidemiological methods have developed greatly, and new ways of handling and interpreting data have been realized. Fortunately, in recent years, systematic reviews known as 'Cochrane reviews' have employed rigorous research criteria for the selection

of papers and methods used. The results are published in full in The Cochrane Library where detailed information can be obtained on which to base decisions on how to cope with the disease problems in the population that dentists are trying to serve.

Randomized controlled clinical trials (RCCT) are the best evidence and in the literature there are a lot of *p*-values based on a statistical test. These tests question to what extent the results obtained might have been achieved by random effects—by chance. Do not be impressed by *p*-values of 0.05 or less. What is important is that one should question what the results mean in terms of the ability to preserve tooth surfaces from developing caries during the test periods. How many more surfaces develop caries in a given period (for instance, 2–3 years) in the experimental group versus the control group? Remember a difference may be 'statistically significant', but does it matter in clinical terms?

It is also important to appreciate that results from a single study are not enough. Cochrane reviews are useful because they gather all the quality studies together and conclude with a 'state of play' of the current evidence. Unfortunately, usually only studies with 'significant effects' are published in the research literature. Negative results are not submitted either because the companies paying for the clinical trials do not want to or journals may be less interested in 'negative results'. Inevitably, if a company has funded the research, they are interested in presenting their 'new compound' as having a 'significant effect', otherwise it won't sell. Companies have a legitimate right to increase their market shares and grow, but 'all that glisters is not gold' from a health improvement point of view. Remember to ask the question—will this serve the need of the population?

Be aware that cost-effectiveness considerations will be strongly influenced by the caries incidence rate in the populations studied. It is important to realize that many clinical studies were conducted when the caries prevalence and incidence was much higher than it is today (for example, in many European countries caries prevalence and incidence has fallen amongst children by as much as 90%). This means it may be very unwise to apply results obtained in one period, in a certain population, to an entirely different population with a very different disease profile.

To give a depressing example of this, some years ago, an international body recommended a country to focus their caries preventive programmes on controlled fortnightly mouth rinsing with fluoride solutions. This had been shown to be very effective in children in several Scandinavian countries in the 1960s and 1970s when the caries incidence rate was still high. The country's sparse resources for oral health care were directed into this programme, but

unfortunately the outcome was not cost-effective. What had been missed was that the caries rate at time of initiation was as low as that in Danish children, where the method had already been abandoned as not cost effective because caries rates had fallen. **There is no one single fluoride method or programme that can be recommended uncritically to all populations**.

4.3.5 Fluoride vehicles

The following section tries not to go into small details, but rather makes some important comments on what the results so far indicate and promotes awareness of some pitfalls when taking a decision on the choice of fluoride vehicle in caries prevention and control.

Water fluoridation

This is highly successful in obtaining caries reduction. For many years, it was about a 50% reduction. In the USA, however, since the late 1980s the difference between populations with or without water fluoridation has diminished to about 15%. In Europe, the best controlled study from Holland, initiated in the middle of the twentieth century, was abandoned in the late 1970s because the difference in caries rates between +F/–F areas had gradually disappeared. Socio-economic conditions change in societies and a multitude of preventive programmes (such as brushing teeth with fluoride toothpaste) operate. Once again, this emphasizes the importance of remembering when the results were obtained. However, it is very important to stress that public water fluoridation is the most cost-effective way of obtaining a caries reduction to everyone in a society in a passive way, by which is meant the individual is not required to actively do anything to benefit.

Fluoride tablets

These have limited, if any, role to play. In most studies, the only effect is that children whose parents comply and give them the tablets develop dental fluorosis. The differences in caries experience probably reflect the difference in mothers' educational background, educated mothers brushing teeth more assiduously and limiting sugar in the diet.

Salt fluoridation

This should not be recommended. Increased consumption of salt should not be encouraged from a medical point of view! Two recent reviews indicated that it might be beneficial for children's permanent dentitions. However, studies examining the effects of salt fluoridation in the prevention of caries are qualitatively of lower levels in the hierarchy of evidence about effectiveness, and data

are, in general, of poor methodological quality. In countries where fluoride has been added to salt for decades, children and young adults show a high prevalence of dental fluorosis.

Fluoride toothpastes, mouth rinses, gels, and varnishes

The series of Cochrane reviews on effectiveness of these are considered comprehensive. In particular they have documented that the daily use of fluoridated toothpaste is the obvious choice from a cost-effective point of view. Moreover, it has been shown that additional caries reductions may be obtained if other topically-applied fluoride treatments (for example, varnishes) are combined with fluoride toothpaste.

It is important to emphasize that it is mandatory that the fluoride in toothpaste is available as free fluoride ion and not bound to the abrasive compounds used in some dentifrices around the world. The fluoride concentration varies considerably from about 1000 ppm to 5000 ppm in some products claimed to be especially useful to adults at high risk of dental caries (for example, those with dry mouth). Although there is a direct correlation between concentration of fluoride in the pastes and the expected caries reduction, this tendency of playing the 'little chemist' in the bathrooms of families is not recommended—in particular not in families with young children, where ingestion at regular intervals over time of a high-fluoride paste used by a child could increase the severity of fluorosis.

For children under 3 years, where swallowing toothpaste risks fluorosis, a smear of a toothpaste containing 1000 ppm F (if available in your country—otherwise use the family paste) on a small brush is suggested. After 3 years, the family paste, 1350–1500 ppm F, is suitable, although in young children only a small pea-sized portion should be used. Avoid toothpastes that are foamy as this will enhance the children's wish to spit it out rapidly—remember it is the elevation of fluoride in the oral fluids, for prolonged periods of time that matters. Even in the elderly, with many exposed root surfaces at risk of caries development—in the approximal spaces as well—this family toothpaste can be used to create a reservoir of fluoride in the oral cavity at night if they apply a small amount on the finger and press the paste into the spaces where cleaning may be difficult. Thereby, they may avoid toothpastes that have a too high—and potentially toxic—content of fluoride. At night the saliva flow almost ceases so an excellent fluoride reservoir effect can be achieved. The detergent (sodium laurel sulphate) used in some toothpastes may, however, cause a burning sensation, particularly in the atrophic oral mucosa of the elderly with a dry mouth. For these patients it will be necessary to find a toothpaste without sodium lauryl sulphate.

Avoid leaving toothpastes with a fluoride content above 1500 ppm lying around because there is a risk that small children may lick or eat the toothpaste! In this context, it is unfortunate that some companies produce particularly sweet-tasting toothpastes for smaller children. As toxicity has reared its head again, this section will end on fluoride with a small section on acute toxic effects.

4.3.6 The toxicity of fluoride—a few comments on taking care

Anyone recommending the use of fluoride-containing dental preparations should be aware of the fluoride content and potential hazards. Over the years there have been many claims that increased ingestion of fluoride may result in a variety of diseases. However, when such claims have been carefully studied, the results have never been confirmed. Recently, in the UK a report has been published in the *British Medical Journal* indicating in an observational study that the prevalence of hypothyroidism is increased in populations exposed to increased fluoride levels in the water supply. It has to be confirmed in other studies and in other populations if such a correlation truly exists and what the biological mechanisms might be.

Information on the acute toxicity of fluoride in humans is gathered from recorded cases of deliberate or accidental overdose. The acute lethal dose is approximately 15 mg/kg body weight, although as little as 5 mg/kg may kill some children. A dose of 5 mg/kg should trigger immediate emergency treatment. Sublethal toxic effects can be produced by as little as 1 mg/kg.

The exact mechanism by which fluoride produces its toxic effect is not known. Symptoms of sublethal poisoning include salivation, nausea, and vomiting. The symptoms usually appear within an hour of ingestion and, if over-dosage appears as a result of topical fluoride application, may not manifest until the patient has left the surgery. Death from respiratory or cardiac failure occurs within 24 hours of a lethal dose.

A small quantity of fluoride (less than 5 mg/kg body weight) is neutralized by drinking a large volume of milk. However, if more than 5 mg/kg has been ingested or if there is any doubt about the exact quantity consumed, the child should be taken to hospital and given gastric lavage. Speed is of the utmost importance, because fluoride is very rapidly absorbed.

Table 4.1 lists some of the fluoride agents in use, the amount required to produce early toxic effects and the potential lethal dose—all in relation to the average 5-year-old weighing about 20 kg. These values would be considerable lower for the average 2-year-old.

Table 4.1 Toxicity of fluoride preparations, calculated for a 5-year-old child weighing 20 kg

	Sublethal acute poisoning dose	Potentially lethal dose
Sodium fluoride varnish (2.26% F)	0.9 ml (1/5 teaspoon)	4 ml (4/5 teaspoon)
0.05% Sodium fluoride rinse	88 ml (4/5 cup)	420 ml (4 cups)
Toothpaste 1000 ppm	33 ml	100 ml
Toothpaste 1500 ppm	22 ml	66 ml
Toothpaste 5000 ppm	6 ml	20 ml

Although no cases of acute toxicity due to ingestion of toothpaste have ever been reported, a 5-year-old could be severely poisoned by consuming about two-thirds of a 100-ml tube of 1500 ppm fluoride paste; a 1-year-old would need to consume only half this amount. Fluoride toothpaste should therefore be kept out of the reach of young children, and this is particularly important with the high fluoride pastes used by some adults presenting with multiple active lesions. Similarly, fluoride mouthwashes should be kept out of the reach of young children.

4.4 The role of diet in caries control

The evidence that the frequency and amount of sugar consumption is linked to caries is irrefutable. Thus, emphasis on diet in caries control would seem logical. Unfortunately, the evidence that it is possible to modify people's diets is lacking! However, in populations where sweets, candies, snacks, and cakes are very common in daily life, these habits will enhance the risk for caries development. A sweet diet may be a country's tradition and some Eastern countries exemplify this. Alternatively, a sweet diet may be introduced and the English sweet tooth was introduced in former colonies. However, since the 'advent' of fluoride, the emphasis in caries control has shifted from diet to oral hygiene with fluoride-containing toothpaste.

However, this does not obviate the dental professional from giving **dietary advice to all patients**. Patients should know that sugar causes dental caries. In particular, patients should be informed that there is sugar 'hidden' in a variety of foods–not only in soft drinks and jam! Manufacturers add sugar to most products, even some meats. In addition, it is almost impossible to open a newspaper today without reading about the global obesity epidemic in children and

young adults. This already has profound implications for health. Being overweight is linked to:

- Heart disease.
- Stroke.
- High blood pressure.
- Type 2 diabetes.
- Some cancers.

Is there a link between caries and obesity? Systematic reviews of evidence tell us there is not, and a link would seem unlikely because, in the developed world, the incidence of obesity has risen, while the prevalence of caries has fallen. Indeed, in some countries, caries is associated with being underweight. Factors such as where in the world you live and socio-economic status affect both caries and obesity, but to link the two would be an oversimplification.

Despite this, reducing the amount and frequency of food intake (currently there is particular emphasis on sugar reduction) would help weight control and reduce dental caries. So this is potentially two benefits (weight and caries) for the price of one intervention and Public Health experts call this a **common risk factor** approach. Dental patients can only benefit from the current emphasis on sugar reduction. In addition, the obesity epidemic is potentially so serious that all health professionals have a responsibility to give advice on diet, in the same way as they have a responsibility to give advice on smoking.

To avoid obesity one should all eat the right amount of food relative to levels of activity. In addition, there are key messages with respect to diet:

- Eat a range of foods.
- Eat fruit and vegetables every day.
- Eat fish every week and in particular oily fish.
- Cut down on saturated fat and sugar. This is not that easy because the content in processed foods is determined by the manufacturer.
- Eat less salt—do not encourage salt consumption. Unfortunately, in processed foods, salt content is determined by the manufacturer.

The consensus recommendations with respect to caries are:

- Drink plain water. Be aware that fruit juices, including those with no added sugar, are cariogenic. Do not add sugar to drinks.
- Reduce frequency and amount of sugars at all meals and between meals.
- Do not eat sweets. Restrict them to preferably 1 day a week;
- Avoid in-between meal snacks such as cakes, biscuits, and sweet soft drinks. Take an apple or carrot instead, and drink plain water for thirst.

Table 4.2 Alternative names for sugar

Beet sugar	High fructose glucose syrup
Brown sugar	Honey
Cane sugar	Hydrolysed starch
Corn sugar	Invert sugar
Corn sweetener	Invert sugar syrup
Dextrose	Icing sugar syrup
Fruit juice concentrate	Isoglucose
Fructose	Levulose
Glucose	Maltose
Glucose syrup	Molasses
Fructose glucose syrup	Sucrose
Glucose fructose syrup	Sucrose sugar
Granulated sugar	Sugar
High fructose corn syrup	Syrup

- Check liquid medicines are not syrups that are sugar based. Products sweetened with non-cariogenic, artificial sweeteners are available.
- Try to obtain a consensus in schools (teachers and parents) not serve sweet soft drinks, sweetened juices, biscuits, and sweets whenever there is a birthday to celebrate.

These damaging sugars are sometimes described as 'free sugars' and are defined as 'mono- and disaccharides' added to foods by the manufacturer, cook, or consumer, and sugars naturally present in honey, syrups, fruit juices, and fruit concentrates. Table 4.2 lists the alternative names for sugar and will be useful when helping patients examine food labels. Foods containing free sugars include the following:

- Sugar and chocolate confectionary.
- Cakes and biscuits.
- Buns, pastries, fruit pies.
- Sponge puddings and other puddings.
- Table sugar.
- Sugared breakfast cereals.
- Jams, preserves, honey.
- Ice cream.
- Fruit in syrup, fresh fruit juices.
- Sugared soft drinks.

- Sugared, milk-based beverages.
- Sugar-containing alcoholic drinks.
- Dried fruits.
- Syrups and sweet sauces.

So is there a recommended daily limit for sugar consumption that is suggested to be commensurate with dental health? A recent review from the World Health Organization (WHO) on the relationship between sugars and dental caries suggested that free sugar intake should be reduced to 5% of a person's total energy intake. Notice how unhelpful this is because it seems impossible to translate this into concrete advice in the surgery! Fortunately, it has been demystified by saying that, for an adult of average body weight, this equates to around **six teaspoons of sugar (25 g) per day**. This is less than one 330 ml can of regular carbonated juice, which contains 26 g of free sugars! Look on the labels of the sweet things you eat. You will be surprised!

Might some government help be needed here to make healthy choices the easy choices? In the UK, manufacturers have reduced the salt content of foods. Comparable legislation to force manufacturers to reduce sugar in food would be an excellent public health measure. Laws to ban smoking in public places have been amazingly successful in reducing smoking. Might something comparable be done for sugar? Your old and grumpy authors are sceptical when it comes to sugar! We look at labels of 'light' products that contain less fat and note the sugar content has been increased! Would increasing the tax on sugary products achieve anything? We doubt this—it has done nothing for alcohol and tobacco consumption. Perhaps dentists could make a real difference if they try hard to suggest that children should drink water between meals and avoid sugary beverages. When asking patients to keep diet sheets, it can be surprising how often a sweet drink, frequently consumed, appears to be an obvious culprit. More of this later!

4.4.1 Sugar and teeth: some questions you may be asked

Is fresh fruit good for teeth?

A healthy diet should preferably contain several (about five) portions of fruit and vegetables per day. The fruit contains intrinsic sugars, but in fruit these are not harmful and the fibre is beneficial to health. Some acidic fresh fruits (e.g. citrus fruit, apples) may cause erosion if eaten on a regular basis in substantial quantities.

Is canned fruit good for teeth?

This can be harmful because the fruit is often in sugar-rich syrup.

Is fruit juice good for teeth?

No, because the juicing process releases the fructose, glucose, and sucrose from the whole fruit. The drink contains less fibre than the whole fruit. Consume only at mealtimes and limit to once per day. 'Fruit juice drinks' should not be confused with juiced fruit. They often contain little fruit and added sugar. Avoid these.

Are fruit smoothies good for teeth?

No, because the blending process releases the free sugar from the fibrous fruit.

Is dried fruit good for teeth?

No, because the dried fruit contains super-concentrated free sugars. It is also sticky so it is not a safe snack.

Is milk good for teeth?

Cow's milk contains lactose, probably the least cariogenic of the dietary sugars. Milk contains the protein casein, which may be protective, but infants should never be put to bed with a feeding bottle containing milk.

Is honey good for teeth?

No, even though it is a naturally-occurring free sugar and may have some anti-bacterial action. Never dip dummies into honey. Never add honey to drinks to make them sweet.

4.4.2 The responsibility of multinational companies

We do not think all multinational companies always consider carefully what they do when trying to sell their products. At the moment toothpastes for children are being announced in advertisements to have 'a sweet and pleasant taste'. Not fermentable sugar—but when tasting, it is so sweet that by using it daily very young children are given an impression that they should seek a sweet taste. They get gradually addicted to sweet taste! No wonder they get obese later. Remember that children should be discouraged from swallowing toothpaste in order not to ingest too much fluoride during tooth development (see section 4.3.2, 'Dental fluorosis'). A 'sweetened' toothpaste from one of the leading companies hardly supports the low sugar message. Interestingly, this was addressed by an obesity expert in a Danish newspaper recently—not by dentists.

It is amazing that there have been so few attempts to truly focus on reducing the total intake of sugar per day. Now the World Health Organization Nutrition Guidance Expert Advisory Group (WHO/FAO) has suggested that the daily consumption of free sugars should be limited to 5% of total calories. This is not the first time the World Health Organization (WHO) and the Food Agricultural Organization (FAO) have recommended limiting consumption of free sugars.

In 2003 they recommended a reduction to 10% of total calories. However, it appears from a recent publication that the World Sugar Research Organization (WSRO) may have successfully blocked their joint recommendations from becoming WHO policy. This is an incredible and disturbing story, and the paper by Kearns, Glantz, and Schmidt should be read (see Further reading).

Who are WSRO? They are a trade organization representing more than 30 international members with economic interests in the cane and beet sugar industry, including the Sugar Association in the USA and Coca-Cola. Thanks to the meticulous work by Kearns, Glantz, and Schmidt, it has now been documented how the sugar industry has allegedly influenced global dental research agendas. They show how in 1971, the National Caries Program of the National Institute of Dental Research (NIDR) was apparently distorted and misleading. Allegedly, they managed to ensure an overlap of the members of the NIDR Caries Task Force Steering Committee and a committee set up in parallel by the International Sugar Research Foundation. In this way, the sugar industry apparently gained direct access to the future National Caries Program policies and research priorities. The sugar industry maintains that it is better to focus on reducing the (unavoidable!) harm of sugar consumption by, for example, advocating fluorides and modification of biofilm composition, adding neutralizing agents to mouthrinses or toothpastes, modifying food products with additives, reducing the virulence of oral bacteria. The end result of this was possibly that limited research has been supported which focuses on **reducing** sugar consumption!

Let this be a warning to all health personnel. This is just one case, but naturally the chocolate and confectionary industries, the producers of soft drinks, the pharmaceutical industry, the growing number of food companies developing food additives and sugar substitutes, will all be trying to influence the market and society with all available means. Let us just predict that as soon as the US market from 2016 gets free access to the EU to advance the sales of high fructose corn syrup (HFCS) as a sweetener, this product will soon become as widespread in EU products as in USA, where it constitute about 20% of the non-sugar sweeteners. This product is produced mainly for economic reasons for use in beverages in the USA. It is cariogenic, although claimed to be slightly less cariogenic than sucrose. Consumers have virtually no chance to check what is written with very small text on the products!!

Unfortunately, too much effort and resources have been spent on caries research without basically obtaining an optimum effect. The position has improved greatly, but with today's knowledge it is possible, using very simple means, to do so much better to control dental caries lifelong—the key points are to limit sugar intake and brush the teeth daily with a toothpaste containing

fluoride! **Dental caries is not a result of fluoride deficiency—fluorides can only modify the rate of mineral loss over time, while sugar consumption speeds up the metabolism in the dental biofilm**. The two approaches of plaque and dietary control must go together for optimum effect.

4.5 Summary of caries control principles

Caries lesion development goes on in most individuals throughout life wherever there is a dental biofilm. The rate of lesion development varies within and between populations depending on dietary habits, socio-economic development, educational background, and the complex interplay between numerous biological variables. Active ongoing demineralization can be arrested at any stage of lesion development if the following caries control principles are followed:

- **Mechanically disturb the formation and structure of the dental biofilm daily!** Use toothbrush, dental floss, and interdental brushes. Make sure all tooth surfaces are included—not just those easily seen in the front teeth.
- **Always use a fluoride containing toothpaste**. Fluoride can help in reducing the rate of mineral loss **when present** in the oral fluids. Brush at least twice a day, spit out, but avoid rinsing with water afterwards.
- **Lower the daily intake of fermentable carbohydrates**. Avoid/minimize sugar intake in snacks, sweets, and soft drinks. Be aware of all the 'hidden' sugar in the food, drinks, and medicines.

Further reading

Carson, S. and Freeman, R. (2014). Fact or fiction: does a relationship exist between free sugars, dental caries and body weight? *Dental Update*, **41**, 862–866.

Fejerskov, O., Cury, J. A., Tenuta, L. M., and Marinho, V. C. (2015). Fluorides in caries control. In: Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management* (3rd edn), pp. 245–276. Wiley Blackwell, Oxford.

Fejerskov, O., Ekstrand , J., and Burt, B. A. (eds) (1996). *Fluoride in Dentistry* (2nd edn). Munksgaard, Copenhagen.

Fejerskov, O., Manji, F., and Baelum, V. (1990). The nature and mechanisms of dental fluorosis in man. *J. Dent. Res*., **69**, 692–700.

Kearns, C. E., Glantz, S. A., and Schmidt, L. A. (2015). Sugar industry influence on the scientific agenda of the National Institute of Dental Research's 1971 National Caries Program: a historical analysis of internal documents. *PloS Med*., **12** (3), e 1001798.

Moynihan, P. J. and Kelly, S. A. M. (2014). Effect on caries of restricting sugars intake—systematic review to inform WHJO Guidelines. *J. Dent. Res*., **93**, 8–18.

NHS Health Scotland (2012). *Oral Health and Nutrition Guidance for Professionals*. NHS Health Scotland, Edinburgh.

Chapter 5

When should a dentist restore a cavity?

At the start of this book it was commented that some dentists see restorative dentistry (fillings) as the **treatment** of dental caries. These dentists see **prevention** of caries as a separate issue. The authors profoundly disagree with this. The previous chapters have shown how dental caries develops and what it is, so in this chapter it is important to ask the question 'with this knowledge in mind, what is the role of restorations (restorative dentistry) in caries control?' Are restorations required or can the problem be solved by sealing all surfaces in the oral cavity—or at least those parts where surface irregularities (occlusal fissures, grooves, pits, etc.) may favour biofilm stagnation? Therefore, this chapter starts with a discussion of so-called fissure sealants.

5.1 Fissure sealants

On occlusal surfaces, caries lesions may form at the entrance to the fissure because this complex morphology may be difficult to clean, particularly in the erupting tooth that is below the level of the arch and tends to be missed as the toothbrush swings by. Fissure sealants cover the fissures with a flowable resin or highly viscous glass ionomer cement, so that they are easier to clean. Their effectiveness has been proved in many studies. When first introduced in developed nations, all molar surfaces were recommended for sealing to avoid caries development and the need for fillings. This 'sealing all teeth' policy would now be totally incorrect for two reasons:

- Caries can be controlled by cleaning alone.
- Many of these surfaces will never develop lesions, and this automatic sealing approach is over treatment and not cost-effective.

5.1.1 Contemporary indications for sealants

The indications for fissure sealing are:

- Active fissure caries has been diagnosed, but attempts at caries control have not arrested lesion progression.
- Occlusal surfaces are often highly irregular, and filled with grooves and fissures, and the patient or parent either cannot, or will not, remove plaque effectively. This is particularly important in the erupting molar. This surface is particularly at risk of lesion development and progression because permanent teeth can take 6–12 months to erupt; indeed, third molars may take several years. During this time, the occlusal surface is below the line of the arch and difficult to keep clean with an ordinary tooth brush.

5.1.2 Materials available

Resin-based materials or high viscosity glass ionomer cements may be chosen. Clinical trials show both are equally effective. The glass ionomer material may be lost from the fissures over time, but this does not prejudice outcome probably because remnants of the fluoride-releasing dental material remain in the depth of the fissures. A glass ionomer material would be specifically selected if there is any anticipated difficulty with moisture control. Exclusion of saliva is essential to the success of the resin material, but glass ionomer cements can tolerate some dampness. For this reason, the glass ionomer material is preferred for erupting teeth and where patient co-operation is a problem. While the resin-based materials are usually light-cured, glass ionomer materials are chemically cured. They would be selected in situations with no conventional dental surgery and no electricity.

5.1.3 Further considerations

Since sealants may be used for initial lesions, where oral hygiene despite advice is poor, it will be inevitable that sealants will be placed over bacteria and sometimes over demineralized dentine. This is of no consequence because the sealed bacteria either die or become dormant with changed metabolism.

Sealants may never stand alone, but should be combined with further caries control education. The patient should view them as a temporary expedient, not an invitation to hand over caries control to the dental team.

Fissure sealants have historically been used on occlusal surfaces, but more recently sealant resins and resins designed to infiltrate the white spot lesion, have been used in non-cavitated approximal lesions. Clinical trials have yet to show consistent success comparable with occlusal sealants. The techniques are clinically demanding, time-consuming, and the materials are expensive. Basically, the whole concept is old fashioned—you cannot keep a mouth free from dental caries by full-crown sealing treatment! Full crowns were often tried in vain in the past, failing because of new caries at the crown margin. Moreover, it has been shown that even deep, non-cavitated lesions can be arrested and further progression stopped (by cleaning and fluoride application) without stuffing the intercrystalline spaces with resins!

5.2 When do we need fillings?

Fillings are required when the patient cannot access the active cavitated lesion with any cleaning aid. Such cavities remain filled with a microbial plaque and are bound to progress. Thus, from a caries control point of view, restorations facilitate plaque control of a given tooth and restore its chewing function.

5.2.1 Occlusal surfaces

Once an occlusal lesion has a cavity, the dentine is always involved in the process. The clinical appearances are described in Chapter 3, Section 3.3, 'Commonly used visual criteria'. These larger lesions are clinically visible in dentine (Figure 3.13f,g and Figure 5.1a), can also be seen on a bitewing radiograph (Figure 3.30 and Figure 5.1b), and can be considered as active. The dentine facing the cavity will have been invaded by microorganisms, and will feel leathery or soft when touched with a probe. The lesion could be arrested by simply fissure sealing because this will cut off the microorganisms from their major source of nutrient from the oral cavity. However, sealants may be lost without the patient realizing this has happened, and the lesion will then progress again. Thus, for the cavitated lesion that is clearly visible in dentine, the predictable procedure is to restore the tooth. Sometimes, all the undermined enamel breaks away and the hole is accessible for cleaning. The lesion would now arrest if

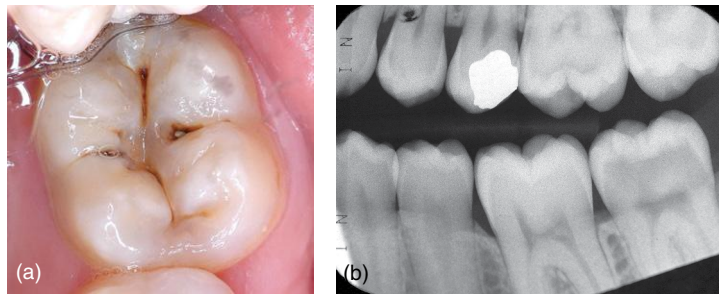


Figure 5.1 (a) Second molar with large, cavitated, occlusal lesion. (b) Radiograph of this tooth showing large lesion in dentine. This tooth is symptomless and vital.

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microbial deposits are cleaned away daily and a filling would not be required. The reader will now realize that an alternative to restoration is to open the lesion for cleaning. This is what happens naturally, as shown in Figure 3.10.

5.2.2 Approximal surfaces

When a cavitated caries lesion is present on an approximal surface, the adjacent tooth may prevent effective cleaning by either a brush or dental floss (Figure 5.2a–c) and the lesion is likely to progress. Approximal surfaces should always be examined carefully in the clinic for the presence or absence of a cavity. However, it may be difficult to see the lesion because it is obscured by the adjacent tooth. If in doubt during a clinical examination then use dental floss. If it catches an irregularity or is shredded and disrupted, it is likely that there is a small cavity. In this case a bitewing radiograph may be useful to detect such narrow and deep dentine lesions (see Chapter 3, Section 3.4.3 'Bitewing radiography', and histogram, Figure 3.31), and assess how close to the pulp they are. However, tooth separation can solve the problem in a few days so that a better overview is obtained (Figure 3.26a,b).

Be aware that the radiograph cannot in and of itself show whether a cavity is present, but a series of unrelated clinical studies have correlated the radiographic appearance to the likelihood of cavitation. Thus, consulting Figure 3.28:

- R4 lesions (inner dentine) will be cavitated and restoration is required.
- R3 lesions may or may not be cavitated. Cavitation is more likely where the patient has several active lesions, where the gingival papilla is inflamed (indicating plaque that the patient cannot remove in the cavity). Tooth separation (Figures 3.26a,b) allows the dentist to feel for a cavity with a probe.

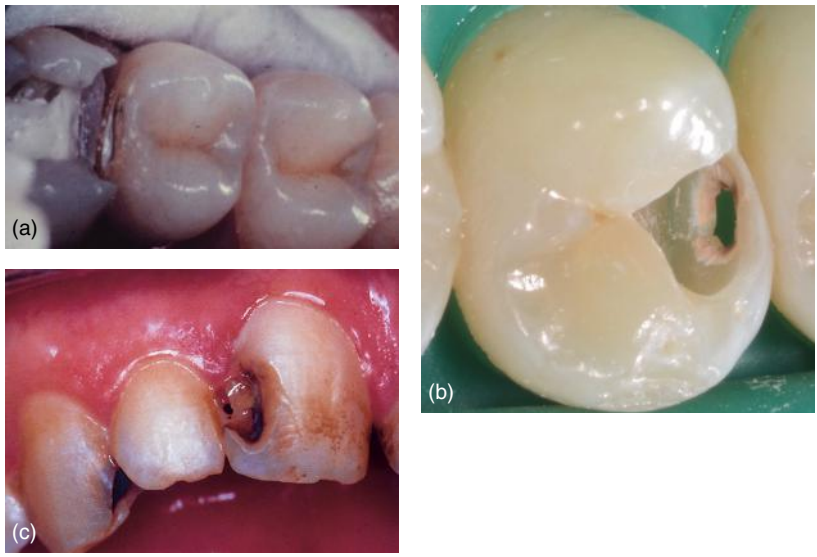

Figure 5.2 (a) Restoration is being prepared on the mesial aspect of the molar. This allows the dentist to see the cavitated lesion on the distal aspect of the premolar. Even the most fastidious of flossers could not clean the cavity; the floss would just skate over the surface. A restoration is indicated to facilitate cleaning. (b) Restoration is being prepared on mesial of upper premolar. The dentist has accessed the lesion, but for the moment has preserved the marginal ridge in order to protect the adjacent tooth from being scored by the bur. The cavity in the enamel can be clearly seen and this is why a restoration is required. Eventually, this enamel will be removed before restoring with composite. (c) The patient finds this lesion difficult to clean and it is unsightly. A restoration is indicated, but the patient should be taught to keep the margins of the filling clean, otherwise a new lesion may form next to the filling.

(b) Courtesy of John Good.

- R2 and R1 lesions are unlikely to be cavitated and it would be quite wrong to place fillings. These lesions should be treated non-operatively and the lesions reassessed for progression or arrest, probably in a year.

5.2.3 Smooth surfaces

In contrast to occlusal and approximal cavities, cavitated smooth surface lesions may be easily reached by a toothbrush (Figure 5.3a), although when the process is undermining the enamel, removing the overhanging enamel by grinding and polishing will facilitate cleaning. This is what has been done in Figure 5.3b. However, despite lesion arrest, notice how ugly these lesions are. In Figure 5.3c,

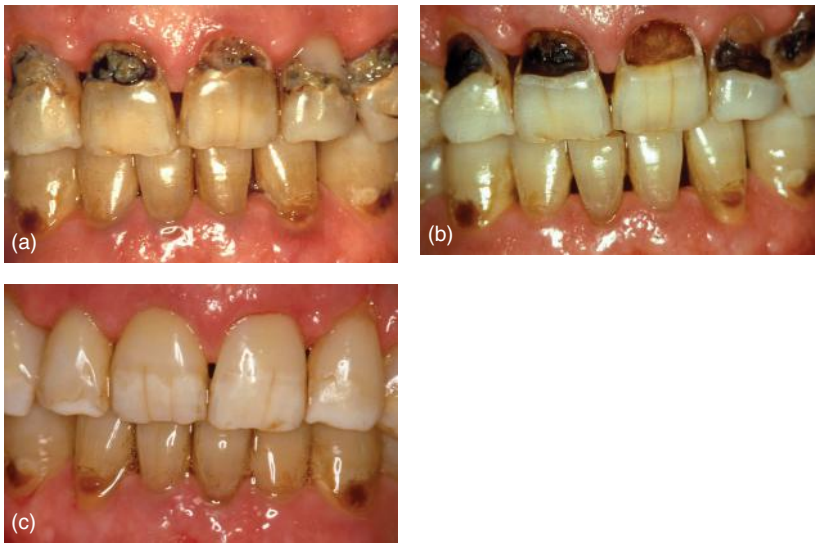

Figure 5.3 (a) Cervical lesions covered by plaque. (b) The same cavities 14 days later after removing overhanging enamel with a diamond finishing bur and giving instruction on cleaning. Teeth were brushed twice a day with a toothpaste containing fluoride. From a cariological point of view, these teeth are now stable, but to improve their appearance they are to be restored with composite. (c) Composite restorations directly after placement. The small colour difference is due to the teeth being dry and this will disappear after some hours when they are wet with saliva.

restorations have been placed and there is a dramatic improvement in appearance. No wonder it is tempting to consider it is the filling that has **treated** the lesion! However, it is essential that patient and dentist realize that the treatment is improving the cleaning, using fluoride-containing toothpaste, rather than the fillings. In distinct contrast to these lesions in visible upper anterior teeth, is the root surface lesion in Figure 5.4a,b,c,d. Over a period of 18 months the patient's cleaning and fluoride application arrested the lesion. The soft, 'infected' dentine was gradually worn away and, finally, the tooth surface is shiny and hard. Although discoloured, this lesion is not visible, even when the patient smiles widely, and for this reason a filling is not required. Indeed, placing a restoration in this circumstance is difficult, even using contemporary adhesive restorative materials. Restoration would not increase the longevity of the tooth, perhaps the opposite, and is best avoided.

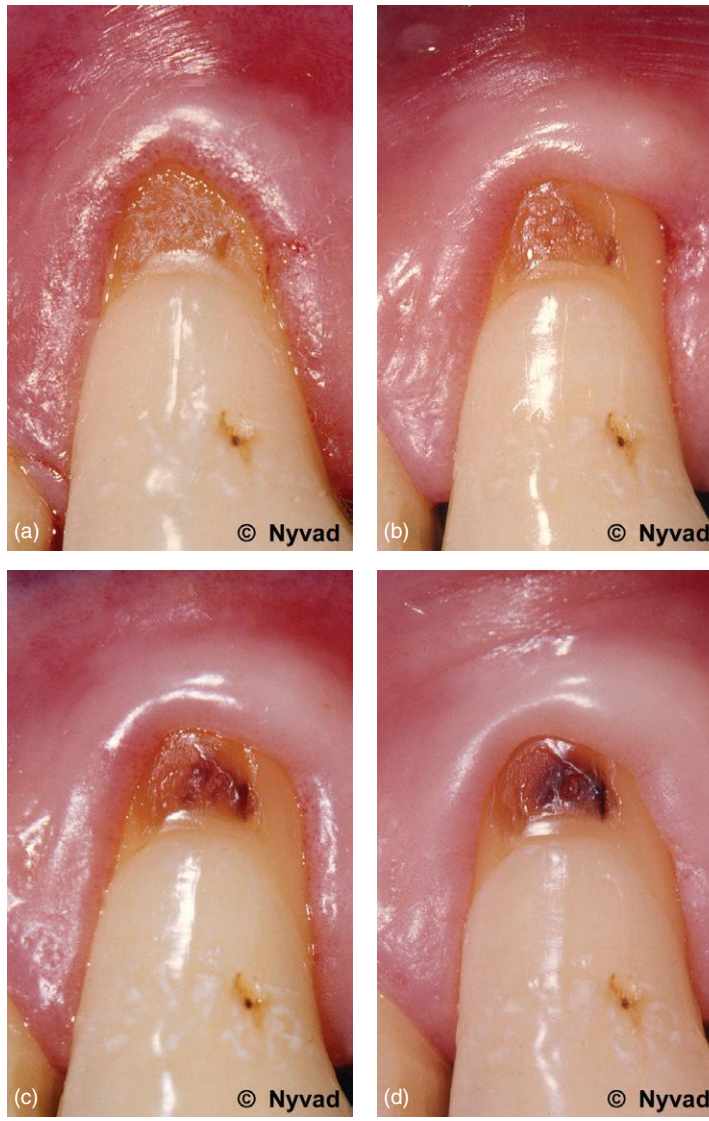

Figure 5.4 Consecutive stages of non-operative treatment of an active non-cavitated root caries lesion on the buccal surface of the upper left canine. The figures show (a) the original appearance of the lesion, then changes in the clinical appearance of the lesion after (b) 3, (c) 6, and (d) 18 months, respectively. Note that within the observation period improved oral hygiene leads to change in colour and surface texture of the lesion, from soft and yellowish to hard and darkly discoloured. Also note changes in the topography of the marginal gingivae.

5.2.4 Replacing restorations

It is salutary to note that some 75% of all operative work is replacement of restorations, particularly restorations made by other dentists, which the new dentist believes can be made better. Why do dentists replace so many restorations? Here is how a repeat restorative cycle is set up, with disastrous consequences:

- Restoration and replacing restorations is considered 'good' dentistry. Restorative decisions tend to be idiosyncratic and aggressive.
- The cause of the lesions is not identified and modified (remember it is the patient who modifies behaviour guided by the dentist).
- Dentists may be paid to fill teeth and not financially rewarded to help patients improve cleaning, diet, etc.
- The more fillings are done, the higher the payment and restorations of mediocre quality are inserted under these time-pressured conditions.
- Decisions about replacing restorations are also aggressive and idiosyncratic, particularly if the patient changes dentist ('I can do this better', may be the attitude of the new dentist). The reasons the restoration is deemed to have failed are not addressed.
- However, cavities increase in size when fillings are replaced—errors in the previous preparations are not identified and corrected.
- Both dentist and patient genuinely believe they are 'treating' the patient and making them healthier.
- As cavities get bigger, the tooth is weakened, the pulp is prejudiced and root canal treatment may be required. Cusps fracture and then teeth are crowned as the ultimate solution.
- Root canal treatment may be inadequate, leaving a nidus for bacterial proliferation and a periapical area develops.
- Eventually, the tooth is extracted, and perhaps a bridge is made or an implant placed.

This depressing restorative sequence is done with the best of intentions, but it is wrong as exemplified anecdotally in the contrast between Figure 5.5a,b. Here, bitewing radiographs of two women, both born in 1944 before the advent of fluoride toothpaste are shown. Figure 5.5a is the mouth of a dentist's daughter whose father told her he could cope with any cavity she produced by placing a filling. She was 50 years old when this radiograph was taken. Caries has continued in this now very heavily restored mouth (the explanation for the continued caries is to be found in Chapter 7, page 153). Many teeth are now unrestorable and this dentition is for the bucket! Figure 5.5b is quite different. This woman was a hygienist's daughter and she was 70 years old when the bitewing was taken. The

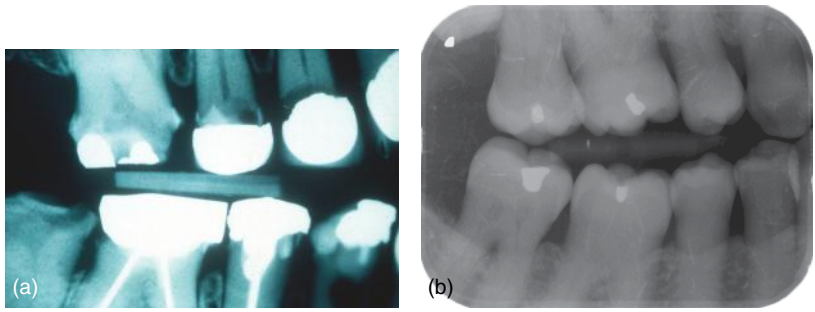

Figure 5.5 (a) Bitewing radiograph of a 50-year-old woman (a dentist's daughter) born in 1944. A very heavily restored dentition is obvious with many new caries lesions around the restorations. Many of these teeth are now unrestorable. (b) Bitewing radiograph of a 70-year-old woman (a hygienist's daughter) also born in 1944. There are very few restorations.

hygienist mother was convinced caries could be controlled by good cleaning and a low sugar diet, and this woman will take a full dentition, with very few restorations, to her grave.

When should restorations be replaced from a cariological point of view? The decision is the same as for primary caries. Cavitated lesions that cannot be cleaned should be repaired or replaced to facilitate plaque control (see Chapter 3, Section 3.1, 'What do we need to know and why?'), but the treatment must also involve caries control measures. When restorations are replaced or repaired for technical reasons (for instance, a fractured or loose restoration, poor appearance, when a restoration is ill-fitting and difficult to clean, open contact point) great care should be taken to define what caused the technical failure so that this mistake is not repeated.

5.3 How 'clean' should the cavity be?

5.3.1 Checks before preparing the tooth for a restoration

Before removing demineralized dentine:

- Ask the patient if there are symptoms such as pain on hot, cold, and sweet stimulae that do not go away in seconds after removal of the stimulus, indicating irreversible pulpitis. A tooth with irreversible pulpitis (note this is a clinical diagnosis) will require removal of the damaged pulp and root canal treatment if it is to be saved.
- Check there is no sinus indicating pulp death. Does the tooth provoke pain if pressed or tapped on?

- Check pulp vitality using cold or electrical stimulation.
- Examine a radiograph. How deep is the lesion? Is there a danger of exposure when preparing the tooth? Does the periapical area show changes that would signify pulp death and the need for root canal treatment.

5.3.2 How is demineralized dentine removed?

The demineralized dentine in an active lesion is soft and wet. It can be excavated using a sharp, spoon-shaped hand instrument (an excavator) or removed with a slowly rotating round bur provided electricity is available. The round bur is ideal for removing soft material from the periphery of the cavity and from the enamel–dentine junction, but a sharp excavator can also be used. Please, avoid air rotors as they destroy too much sound tissue. Restorations need a good cavity seal, and the sound tissue at the enamel–dentine junction and/or the periphery of the cavity if this is on dentine, is essential to achieve this bond with the restorative material.

5.3.3 How much soft dentine should be removed over the pulp?

This is a contentious area, where dentists have been disagreeing for at least 100 years! Should all 'infected', soft dentine be removed and excavation continued to hard dentine, irrespective of the fact that the pulp may be exposed by this, or should carious dentine removal be more selective and superficial to avoid pulp exposure? Thanks to 50 years of careful clinical experiments, it is now known that selective caries removal, leaving soft dentine in a deep cavity, is preferable provided the tooth is symptomless and vital. Indeed the evidence for this is now so overwhelming **that a non-selective, i.e. a complete removal with the risk of pulp exposure should be considered unethical.** (Note: in the literature, excavation to hard dentine may be referred to as 'complete caries removal', while the more superficial excavation to avoid exposure in deep cavities, has been called 'partial caries removal'.)

A number of biological reactions underpin this approach (see Chapter 2):

- The two defence reactions of tubular mineralization (seen histologically as translucent dentine) and tertiary dentine reduce the permeability of the dentine, walling off the pulp from the bacteria invading the dentine in the lesion.
- Once a restoration is placed that seals the cavity, any remaining bacteria either die or change to reflect a non-cariogenic flora that can survive in the stressful (for them!) conditions of a lesion sealed from the mouth by the filling and from the pulp by the biological defence reactions.

- Even if the tooth is excavated aiming to remove all bacteria, this will not be achievable because some of the bacteria will survive, however hard the tooth is scraped, although the bacteria will probably be killed with well-sealed restoration!

Stepwise excavation

Stepwise excavation involves removing only part of the soft dentine caries at the first visit (Figure 5.6a). The cavity is temporarily restored and re-opened after a period of weeks (Figure 5.6b). Further excavation is now carried out prior to definitive restoration (Figure 5.6c). The idea is to arrest lesion progression and allow the formation of tertiary dentine before final excavation, making pulpal exposure less likely. The technique has been extensively investigated (some 24 studies with four randomized controlled clinical trials), in deciduous and permanent teeth, with the following results:

- Clinical success was high and exposure usually avoided.
- Where there was 'complete' caries removal in a control group, in up to 53% of cases, the pulp was exposed.
- Different materials have been used for temporary restoration, including temporary cements, amalgam, glass ionomer cement, and composite. The material seems less relevant than the permanence of the filling. Where fillings were lost, teeth gave symptoms.
- On re-entry (after 25 days to 2 years) the dentine was changed, it was harder, dryer, and darker (Figure 5.6b).
- Microorganisms were cultured in some studies and numbers were hugely reduced on re-entry and those remaining were no longer the acid-producing, cariogenic flora.
- Subsequent excavation exposed the pulp in 17% of teeth.

So is re-entry for further excavation needed? Clinical trials continue, but there is no definitive answer, and **the authors would not re-enter**. The second visit seems expensive, time-consuming, and potentially damaging for no proven reason at the moment. Please note, all research on this topic must be done clinically. There can be no pulpal response in the laboratory making results meaningless.

Selective caries removal and indirect pulp capping

In these studies, there is selective caries removal and a permanent restoration is placed at once. **Indirect pulp capping** is also a selective caries removal technique, but aims to remove all but the last sliver of soft dentine before restoration. This sounds fine until put into practice and the difficulty of knowing where the last sliver and the pulp are becomes apparent. This results in guesswork

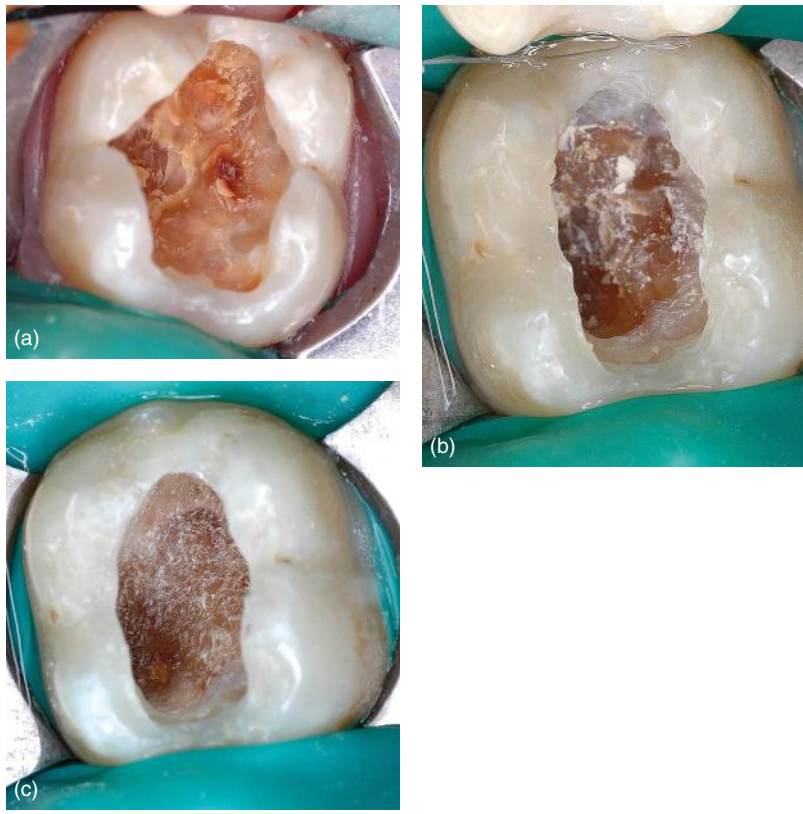

Figure 5.6 (a) The clinical and radiographic appearance of this tooth can be seen in Figures 5.1a,b. The tooth is symptomless and vital, but the lesion is deep and vigorous excavation may expose the pulp. The enamel overlying the lesion has been removed with an air-rotor and the enamel–dentine junction has been made hard by removing demineralized dentine with a slow speed round bur. The top layer of soft, wet dentine has been removed with an excavator, but the dentine remaining is still soft and wet. The lesion is now restored with a composite restoration. (b) The dentist has removed most of the composite restoration after some weeks and the dentine is now dry, darkly coloured, and harder. (c) This harder dentine has been further excavated by scraping it with a sharp excavator. The tooth is now permanently restored with a bonded composite restoration.

and causes nervousness in the process! Since the results of a more conservative removal are good, this is the preferred approach. Figure 5.7a–f shows indirect pulp capping in a deciduous molar. The demineralized dentine over the pulp has been gently removed with a rotating brush and fluoride toothpaste.

Results from selective caries removal studies are good. Teeth survive pain-free for years. There is no current evidence that the fillings collapse or the

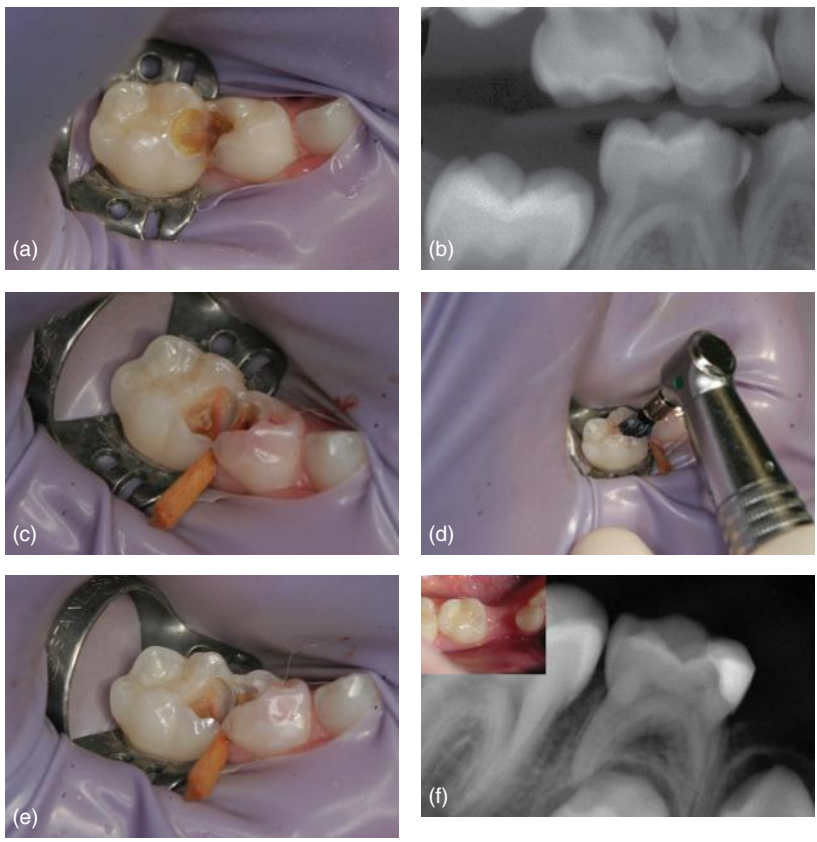

Figure 5.7 (a) Second deciduous molar with deep carious lesion before indirect pulp treatment. (b) The same tooth on bitewing. (c) After excavation of the dentine-enamel junction. (d) Removing very soft, infected dentine with a rotating prophy brush and fluoride tooth paste only. (e) The completed, dried cavity. A resin-modified glass ionomer liner (Vitrebond/3M Espe) was applied and the cavity was restored with a compomer (Dyract/Dentspky Caulk). (f) Clinical and radiographic result after 2 years and 4 months.

Courtesy Rene Gruythuysen and Tandartspraktijk, BSL, Springer Media, Houten, the Netherlands.

bond between the filling and the tooth suffers from this approach. The dentist should be aware that the residual caries will show, as a dark shadow beneath the filling, on a subsequent radiograph. The caries removal approach should be discussed and agreed with the patient, and what has been done must be recorded in the notes. Remember if the patient goes to another dentist they might remove your filling because demineralization (residual caries) is present on radiograph.

Whatever happens, remember to inform the patient about why and how the treatment is planned, and keep a careful clinical record! If the patient decides to seek another dentist one day, offer the patient a copy of their record, which may be given by the patient to the new dentist.

5.4 Managing deciduous teeth

The caries process is the same whether teeth are deciduous or permanent. However, the following are relevant to managing deciduous teeth:

- Deciduous teeth are temporary, only in the mouth for 6–9 years.
- They are smaller with broader contacts, and the pulp chamber proportionally larger relative to the size of the crown. These dimensions means it takes less time for the lesion to reach the pulp in deciduous compared with permanent teeth.
- Loss of the deciduous second molars may cause crowding of the permanent teeth.
- Their owners are immature.
- Frightening the child can have serious consequences for subsequent dental care.
- Pain in children is particularly distressing for parents, as well as children.

Is restorative care useful while the poor dental behaviour that caused the need for fillings persists? Might it be better to achieve excellent cleaning before any operative treatment other than pain relief? It is interesting that the results of full restorative care under general anaesthesia are disappointing. This is probably the best example of the dentist taking responsibility for solving the problem by mending the teeth. However, new caries lesions develop in a few years' time and this is possibly predictable because caries control in the young child must rest with the parent.

5.4.1 Care options for cavitated deciduous lesions

Pain is always managed first. Teeth with irreversible pulpitis do not settle after caries removal, but require removal of the pulp or extraction of the tooth.

Cavitated lesions in pain-free teeth that are not cleansable will progress and something more is needed if pain is to be prevented. These lesions may show transient pain with hot, cold, and sweet stimulae (these are symptoms of irreversible pulpitis). The management possibilities are:

- No caries removal, but 'open' the lesion to allow cleaning.
- 'Sealing' the entire carious tooth.
- Selective caries removal and restoration.
- Non-selective (complete) caries removal and restoration.

No caries removal, but open the lesion to allow cleaning

This approach is often referred to as **non-restorative cavity treatment** (NRCT) because no filling is placed. The rationale is to open the cavity to make it accessible for plaque control, rather than mask the caries activity by filling the tooth. When the cavity has been opened, fluoride varnish (or a 2% sodium fluoride solution) is applied to the carious dentine or, in a deep and sensitive lesion, a layer of glass ionomer cement is placed on the cavity floor. It may take more than one visit to open a tooth sufficiently, depending on the co-operation of the child. The technique is shown in Figure 5.8. The parent is shown how to clean away the dental biofilm regularly every day with fluoride-containing toothpaste. In time, the lesion will arrest and the deposition of mineral within the tubules and

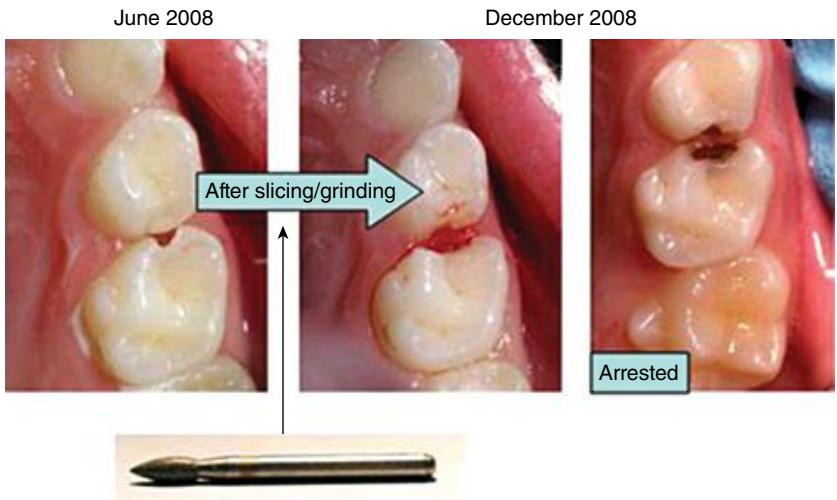

Figure 5.8 A bullet-shaped diamond bur is used to slice and grind the approximal surface so the lesion is opened for cleaning. After 6 months the lesion is arrested—it is darker and harder.

Courtesy Rene Gruythuysen.

tertiary dentine is encouraged. Both processes will decrease tooth sensitivity. It is claimed this management is child friendly and tolerated by very nervous patients, without the need for local anaesthetic, and that several teeth can be 'opened' for cleaning in about 10 minutes.

The technique puts the responsibility for caries control with the parent. It is not a case of 'I will cope with your child's caries by filling the tooth', rather 'You can control caries and avoid a pain reaction by keeping these teeth clean'. Even if the parent's compliance is not perfect, it is claimed the technique slows down lesion progression so that the teeth may survive until shed. Slowing the process gives time to change parental attitudes. Counselling and/or motivational interviewing is an essential part of the technique.

'Sealing' the entire carious tooth

In 2006 a new technique for sealing cavitated lesions with stainless steel crowns was described, which seemed to break every rule in the restorative book! A stainless steel crown was cemented, with glass ionomer cement, onto a cavitated, symptomless, vital, deciduous tooth with no local anaesthetic, no caries removal and no tooth reduction (Figure 5.9a,b,c). The cemented crown was inevitably 'high on the bite' but in 2–4 weeks the occlusion was re-established. This technique is called the **Hall crown** after the Scottish practitioner who devised it. Clinical trials have shown excellent results. The crowns take the teeth symptomless to exfoliation and are preferred by the children to conventional restorations. This is a very different approach to the traditional paediatric dentistry textbook recommendations!

Figure 5.9 (a) Lower right E before fitting crown. (b) Hall crown directly after cementation. (c) Six weeks later the bite has nearly re-established.

Reproduced from *Dental Caries: The Disease and Its Clinical Management,* 2nd edn, Fejerskov, O. and Kidd, E., copyright (2009) with permission from John Wiley and Sons. Courtesy of Nicola Innes.

Selective caries removal and restoration

This has already been discussed in this chapter in Section 5.3.3, 'How much soft dentine should be removed over the pulp?'.

The atraumatic restorative technique (ART) should also be considered. This was originally developed to meet the need for restorative treatment in deprived areas, where there was neither electricity nor running water, but the technique is also of benefit in the conventional surgery setting. It is less stressful and less painful for the child than the traditional restorative treatments and local anaesthesia is not usually required. This makes the technique very suitable where there is dental fear and behavioural problems.

Demineralized dentine is removed with hand instruments and cavities restored with a high-viscosity glass ionomer cement. The adjacent fissures are sealed with the same material. Occlusal restorations are usually successful, but approximal restorations are prone to failure by loss or fracture of restorations because the glass ionomer material may not be strong enough in this situation. Restoration loss is not necessarily a failure provided the remaining hole in the tooth is kept clean.

Non-selective (complete) caries removal and restoration

Many dentists still favour this approach, but it can be demanding of the child and the dentist, involving local anaesthesia, use of high speed handpieces, and good moisture control. Small lesions are managed by intracoronal restorations, but larger lesions are restored with stainless steel crowns having carried out the necessary tooth reduction.

5.4.2 What factors are relevant to choice of management?

The following factors are relevant to the choice of technique:

- **Where in the world are you?** If the child can be seen again soon and the parents are responsive to their role in caries control, use the least harmful approach and observation (remember that dental caries does not develop overnight). Do not just focus on the carious tooth, but the entire dentition. Do the parents (and even the child) understand their responsibility? This must be checked on recall.
- **If a restoration is required and a conventional surgery is not available**, ART techniques will be chosen.
- **Pain might make extraction preferable**. The demeanour of the child should also influence decisions—is this child fragile or robust?
- **The size of the lesion and the age at presentation**. The younger the child presents with lesions, the worse the outcome.
- **Avoiding pain/discomfort**. Some dentists consider techniques avoiding local anaesthesia (e.g. NRCT, ART, and Hall crowns) are helpful when dealing with

nervous patients and may avoid treatment-induced anxiety in the first place. The sedation approach is a short-term expedient for getting the job done, but does nothing to help the patient overcome fear in the long term.

- **All these approaches must be combined with encouraging the parent to take responsibility for caries control (tooth cleaning and diet)**. The open for cleaning approach does this in a particularly obvious way.
- **It may be possible to combine techniques in the same patient**. For instance, occlusal lesions may be managed with ART glass ionomer restorations, while larger, approximal restorations (these tend to fail because the material may not be strong enough in this situation) may be better managed by opening for cleaning or, alternatively, fitting a Hall crown.
- **The time taken in the various techniques is important because time is money**. There seem to be no studies that have specifically examined this.
- **Dentists' fees**. What is remunerated? Caries control measures are often not remunerated, which is ridiculous as this is what matters most!
- **Skill of the dental team**. Many of these procedures may be carried out by trained oral health care persons other than dentists, but under supervision (note legal aspects).
- **How many children are on the waiting list?** Time must be shared sensibly.
- **Views of patients and parents**. Perhaps this is of overriding importance, but is rarely assessed in research, although the research on the Hall technique is a notable exception. Finally, look at Figure 5.10a,b and the questions posed. We do not know the answers!

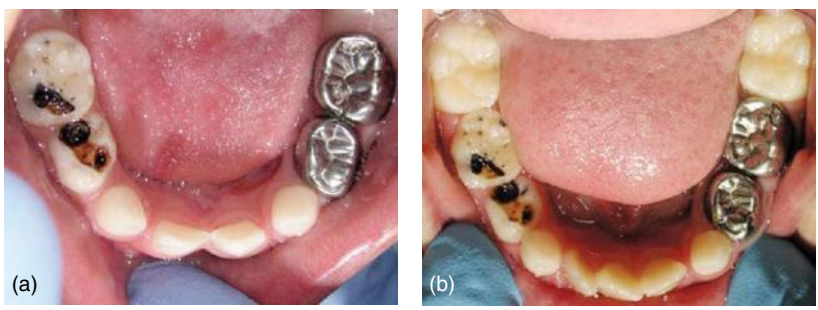

Figure 5.10 Stainless steel crowns on one side of the arch and the NRCT approach on the other. The lesions that were opened for cleaning lesion are arrested. There is 14 months between these pictures and, in that time, the first permanent molars have erupted. There are no lesions in these teeth. **Question**: Which technique is easier for the child, the parent? Which technique does the reader think sends the message to the parent that they are in charge?

Courtesy Rene Gruythuysen and Tandartspraktijk, BSL, Springer Media, Houten, the Netherlands.

5.5 Managing permanent teeth

Each of the management options for deciduous teeth can be used for permanent teeth, but since these teeth are in the mouth for decades, the dentist is likely to choose a permanent restorative material, paying particular attention to the following.

5.5.1 Conventional surgery setting

- **As little tooth structure as possible should be removed to preserve the strength of the remaining tooth**. This will be facilitated by the use of adhesive materials (composites and glass ionomer cement).
- **Take care to protect the adjacent tooth when preparing an approximal restoration**, particularly when using an air rotor. It is all too easy to score it and this will make new lesion formation on this surface more likely. Take care not to overcut when using an air rotor!
- **Establish a contact point**. This can be technically difficult.
- **The margins should fit, which is a challenge cervically**. Unfortunately, this is the area that is most difficult to clean, and a ledge in this position will encourage plaque stagnation, causing a new lesion formation.
- **When deciding to replace a restoration, be very clear as to why this option has been chosen**. There are two reasons—new caries or technical failure of the previous restoration. New lesions are a failure of caries control, and cleaning and diet should be checked. Technical failure is challenging. The dentist who has not introduced exactly the same fault when trying to correct something is either myopic or delusional!
- **Always consider whether the tooth could be repaired, rather than replacing the restoration**. This will conserve tooth structure and in the long run it may be safer for the tooth.

5.5.2 No conventional surgery available

The ART technique was originally developed for use in underdeveloped countries, where no conventional surgery was available. Basically, there were several similar techniques and approaches introduced in different African populations, but where no electricity is available, hand instruments and glass ionomer materials were gradually shown in many populations around the world to be an appropriate choice.

It is now obvious that the approach also has invaluable applications in high income countries. Further clinical trials should be encouraged. For instance, perhaps the patient is living in residential care, and attending a surgery

would be both difficult and stressful. The technique is perfectly suited to domiciliary care.

5.6 Is minimal intervention the solution to restorative dentistry?

You will hear much about minimal intervention dentistry. It stresses a preventive, caries control philosophy where diagnosis of active lesions is followed by energetic, non-operative care and reassessment. The aim is to maintain healthy teeth for life and minimize the need for operative intervention. This chapter has been about minimizing restorative dentistry.

However, when restorations are required, interventions should be as conservative (minimally invasive) as possible preserving as much of the natural tooth as possible. Cutting a tooth will inevitably weaken it and start a restorative cycle where it is all too easy to replace restorations. Adhesive materials and repair of restorations, rather than replacement will help to preserve tooth structure. However, note that minimal intervention easily becomes just a new buzzword for doing what dentists are basically trained to do—drilling and filling. Restorative intervention—irrespective of size—will always be the start of a 'restorative cycle'. The cycle may just take longer and involve more visits to the dentist if the concepts of caries control are not emphasized!

Finally, a word of warning. Is minimal intervention dentistry the key to changing the emphasis of the profession, and those who pay them, from restorative dentistry to caries control? This seems logical and yet, at the time of going to press, there are no realistic fees in England and Wales for caries control measures in the Health Service. Hygienists often work privately and yet caries is concentrated in socially-deprived people who cannot afford private fees. The current system seems utterly wrong, but the authors think it is worse than that—it is unethical. New systems are piloted, but basically the professional attitude remains with a focus on 'smart' technical solutions. Perhaps it is time to reconsider. See the Epilogue and judge whether the authors are just old and grumpy, or whether there is a point here!

Further reading

Banerjee, A. and Watson, T. F. (2015). *Pickard's Guide to Minimally Invasive Operative Dentistry* (10th edn). Oxford University Press, Oxford.

Elderton, R. J. (2003). Preventive (evidence-based) approach to quality general dental care. *Med. Princ. Pract.*, **12(Suppl.)**, 12–21.

Frencken, J. E., Peters, M., Manton, D., Leal, S. C., Gordan, V., and Eden, E. (2012). Minimal intervention dentistry for managing dental caries. A review. *Int. Dent. J.*, **62**, 223–243.

Kidd, E. A. M., Frencken, J., Spleith, N., Opdam, N., and Nyvad, B. (2015). Classical restorative or the minimally invasive concept? In:Fejerskov, O., Nyvad, B., and Kidd, E. A. M. (eds) *Dental Caries. The Disease and its Clinical Management*, pp. 335–374. Wiley Blackwell, Oxford.

Kidd, E. A. M., Bjorndal, L., and Fejerskov, O. (2015) Caries 'removal' and the pulpo-dentinal complex. In:Fejerskov, O., Nyvad, B., and Kidd, E. A. M. (eds) *Dental Caries. The Disease and its Clinical Management*, pp. 375–386. Wiley Blackwell, Oxford.

Qvist V. (2015) Longevity of restorations. In:Fejerskov, O., Nyvad, B., and Kidd, E. A. M. (eds) *Dental Caries. The Disease and its Clinical Management*, pp. 387–404. Wiley Blackwell, Oxford.

Chapter 6

Communicating with the patient and trying to influence behaviour

6.1 Who controls dental caries?

In the previous chapters, the point has been made that dental caries is controllable by the patient regularly disturbing the biofilm, the use of fluoride, especially in toothpastes, and a sensible, but not draconian diet. The success of these strategies depends on the patient, but patients may choose not to comply with the health advice given to them. Many know they should not smoke, should lose weight, and take more exercise, but choose not to alter their behaviour. Altering a patient's behaviour may be key to caries control, and for this reason all members of the dental team should be interested in strategies to modify behaviour.

6.2 Motivation

Motivation is about unlocking the desire within another to make a useful change in behaviour. Good communication is one of the foundations for motivation. Compliance is not likely where patients do not understand, or cannot remember the message. However, people do not change their behaviour just because someone tells them, however clearly, that this is a good idea. Motivation comes from within and cannot just be instilled. It should also be remembered that motivation to change is something that comes gradually, with most people feeling ambivalent about change. Someone who is ambivalent may see a reason to change, but may also see a reason not to change. When we try to persuade someone who is ambivalent to change, the danger is they will resist, giving voice to the counter-argument as to why they cannot change. Actually, the best way to achieve change is if the patient, rather than the health professional, says why and how they should change. In other words, it is their idea and we are there to support it. Despite all these difficulties, good communication can make all the difference in achieving behaviour change and, for this reason, this chapter will now take a detour to discuss aspects of communication.

6.3 Communication

Communication is made up of more than just the actual words used to convey information. The tone used conveys the speaker's emotions and attitudes, and so-called non-verbal communication or body language can be just as important as the actual words.

6.3.1 Actual words

Use of open and closed questions

Open questions encourage patients to respond and discuss their concerns. The patient's concerns are the key that unlocks the approach to this particular patient. To give an example of an open question: 'There are many holes in your teeth; how do you feel about this?' This question encourages the patient to talk, to give their opinion. Whereas the question: 'Are you concerned about the number of fillings I suggest?' is an example of a closed question where a simple 'yes/no' answer will suffice.

Dental jargon

Patients may misunderstand common dental words such as 'biofilm/plaque', 'caries', and 'bacteria', and we may need to check that we are being understood. The words chosen will vary according to the knowledge and

expectations of the listener. To give an example, the cause of caries will be explained differently to a chemistry graduate in comparison with a child. The listener should not be belittled or befuddled by the message.

Listening and empathy

It is important to listen to what the patient has to say. It can be good to repeat or paraphrase what they have said, so it is absolutely clear their message has been taken on board. Empathy refers to the feeling that the listener is making an effort to understand the situation from the speaker's point of view. 'So what you would really like is…'.

Forgetting and distortion

It is easy to forget information, especially if the situation in which it is given is a stressful one. The surgery is a stressful place for many. Alternatively, rather than forget, the patient may distort the message, because it can be difficult to remember correctly. For example, if the patient agrees to try a new toothpaste it would be sensible to write down what has been agreed and/or make a follow-up phone call to make sure it has proved possible to find and use the toothpaste suggested. This follow-up shows care and concern.

Tone

Tone conveys attitudes and emotions, such as enthusiasm or boredom. Patients will soon detect these emotions from our tone of voice and, while enthusiasm is infectious, boredom is demotivating. Similarly, we may pick up signals from the patient's tone.

6.3.2 Non-verbal communication or body language

Non-verbal communication, sometimes called body language, is very important in conveying emotions and attitudes.

Facial expression and eye contact

One of the main functions of facial expression is to communicate emotions and attitudes. Patients will pick up facial expressions so in order not to register judgement, disapproval, and disbelief, or even dislike, take care! Conversely, facial expression can enhance communications, and something as simple as a smile helps to put patients at their ease and enhance trust. It is possible to pick up the patient's attitude from their facial expression, so eye contact is important. Make sure when talking to the patient that the chair is upright, so you and your patient are at the same level and can look at each other. Do not try to have a conversation with the patient lying down in the dental chair.

Body posture

Posture can communicate as clearly as words. If you are running late and the patient is in the waiting room with hands on hips and chin thrust forward this communicates a warning to proceed with caution! When running late it is wise to inform the patient of the delay and apologize for it. Starting with an immediate apology will usually defuse a potentially explosive situation.

Body contact and spatial behaviour

Body contact in our culture is controlled by strict rules, and is used mainly by families and courting couples. Medical professionals use it, but not as a social act. Patients usually accept that their visit will involve touch, but it should be remembered that bodily contact can help or hinder communication. A hand on the shoulder of an anxious patient as the chair is taken back may convey care for the patient and reassure the anxious, but a confident patient may not like it. Each case needs to be assessed individually.

In most walks of life the importance of personal space is recognized and respected, apart from situations where overcrowding is accepted as normal, such as lifts and crowded public transport. Four spatial zones have been suggested:

- **Intimate zone**: 15–50 cm.
- **Personal zone**: 0.5–1.2 m.
- **Social zone**: 1.2–1.6 m
- **Public zone**: 3.6 m.

The personal space referred to as the 'intimate zone' is usually reserved for close friends, but it is automatically invaded when examining or operating on the patient. This is accepted by patients, but it is worth remembering that for some patients there may be some embarrassment and anxiety that affects them receiving communication in this intimate zone. Just because it is culturally acceptable, it should not be taken to be without meaning for the patient. So to discuss with the patient, return the chair to the upright position, ensure eye contact on the same level and move away from the patient to a position where communication is comfortable for both parties.

Clothes and appearance

Clothes and appearance, and the style and décor of the practice and the surgery, all communicate a message to the patient. The best advice given to a group of new students was that when you walk through the waiting room, the patients should hope they might be seen by you and not be thinking 'I hope that is not my student'. Try to look at your surgery and waiting room objectively. Is it covered in dirty peeling paint and old posters?

6.3.3 Communicating the message

There will undoubtedly be times when you need to communicate a message, although do not kid yourself that all you have to do is tell the patient what to do and this will motivate them to change their behaviour. However, just because knowledge alone will not prompt behaviour change, do not assume the patient does not need to know what they should do and why. What factors aid this communication?

Amount of information given

Brief interventions, with a small amount of information, often work better than long explanations. Exploration of the literature on this will show that 'brief' can be a short as 1 minute or as long as 45 minutes! Obviously, time and the amount of information given will depend on what you are doing. To check the fluoride content of your patient's toothpaste is rapid, provided they have the paste with them. On the other hand, if they have gone to the considerable trouble of keeping a diet sheet, you should allow 30 minutes for analysing problems and, above all, listening to them.

When you are asked for advice, make it short, simple, and specific and, if appropriate, write it down. Perhaps a parent has asked what paste and brush they should buy as the first tooth erupts. Show them exactly what you suggest—it should be to hand in the surgery.

Sometimes it will be necessary to check that what you have said has been understood. Perhaps you have agreed with the patient that they will use an interdental cleaning aid in one position in the mouth. Do not just talk about it—ask them if they would be so kind as to show you. Then you know they have the correct toothbrush or floss, know where to position it and can access it. Perhaps, despite everybody's best endeavours, they just cannot do it. Then you must think again.

Making use of other senses

Words are not the only way to communicate. Sight, sound, touch, taste, and smell can all be used, and it is not difficult to involve senses other than hearing. For instance, when teaching a toothbrushing technique, place a hand over the patient's hand to guide their movement. The patient will **feel** the brush touching the gingivae, and if you suggest a scrub on an occlusal surface, they will **hear** the brush. Alternatively, if you suggest the use of the Bass technique, perhaps cervically, effective insertion of the filaments and the vibratory movement is silent. If there is a scrubbing noise, they may be doing it wrongly.

When brushing, the patient may **see** blood on the filaments of the brush or on the dental floss. This is important because some patients regard this as normal,

but would be horrified if their fingers bled when brushing their nails. The patient needs to know that bleeding indicates gingival inflammation as a consequence of poor plaque control. This can be eliminated by correct cleaning and it is important to make the patient aware of this. Knowledge alone will not change their behaviour, but without knowledge, why should they even consider changing?

The patient may also be encouraged to run their tongue over their teeth to **feel** the shiny smooth feeling of plaque-free teeth. When flossing, the patient may feel calculus obstructing the smooth passage of the floss and may also **smell** the putrid odour of plaque removed from a stagnation area. With your help, the patient may become dentally aware of what is happening in their mouth. You are giving them the knowledge that you have.

A telephone reminder

A telephone call (or sms, or e-mail), to show interest in the process of change and support their efforts may be helpful. It can be particularly useful to remind a patient of their appointment and if they need to remember to bring something essential, such as a completed diet sheet.

6.4 Changing behaviour

This is the nub of the problem. Health professionals have traditionally used a biomedical model of disease to target illness through preventive and educational approaches. The assumption is that once the individual acquires the relevant knowledge and skills they will alter their behaviour to maintain optimum health. However, this approach has been shown to be largely ineffective because it overlooks the context that determines human behaviour. This context includes the social, economic, political, and environmental circumstances of the client.

There are models for health behaviour modification. To give examples:

- The **health belief model** that suggests that for an individual to take action they need to believe they are susceptible to the disease.
- The **health locus of control model** that suggests individuals hold beliefs as to whether they have control over what happens to them. An external locus of control implies a fatalistic approach—'my teeth will rot and there is nothing I can do; it is the dentist's job to control disease'. The opposite, internal locus of control, will be the patient who believes they can influence the process.
- The **transtheoretical model of behaviour change** describes the patient's readiness for change. This assumes that behaviour change is a dynamic, non-linear process involving district stages. These stages are:
  - **Pre-contemplation**: not even considered change.
  - **Contemplation**: considered change.

- **Preparation**: getting ready to change.
- **Implementation**: changing.
- **Maintenance**: keeping it up!

This last model provides a framework for understanding the process of change. The technique called **motivational interviewing** is a means of facilitating this process. In this technique the professional does not assume the expert role, but places the client in the role of the expert. The client decides how to interpret the information, and whether to use it in the context of their own life and social circumstance. The professional tailors the intervention to suit the patient's needs and their readiness to change. The next part of this chapter will focus on the technique of motivational interviewing (MI).

6.5 Motivational interviewing

MI has been defined as 'a client-centred directive method for enhancing intrinsic motivation to change by exploring ambivalence'. Let us unpick and explain this definition:

- 'Client-centred' means the client is cast in the role of the expert—their views and beliefs are the essential starting point.
- 'Directive' implies that the professional will take deliberate steps to facilitate a particular behavioural outcome.
- 'Enhancing intrinsic motivation' acknowledges that motivation is not something that can be given; it must come from within the client. It is the internal motivation for change that is encouraged, rather than changes being imposed externally.
- 'By exploring ambivalence' warns that the client may be ambivalent about change, seeing reasons for and against it. It will be important to acknowledge and explore these attitudes.

6.5.1 History and evidence base

MI was originally developed for the treatment of addictive behaviour, particularly alcohol addiction. Results from its use in smoking cessation were originally not encouraging, but more recently, its use by family physicians has shown the technique can increase smoking cessation. Of relevance to oral health is its effect on dietary habits, where it has encouraged changes in overall intake, fat and carbohydrate consumption, cholesterol and salt intake, and consumption of fruit and vegetables. Some studies have focused on oral health, especially the prevention of early childhood caries.

6.5.2 Key principles of motivational interviewing

There are four underlying general principles:

- **Express empathy** for the patient's behaviour change dilemma. This means the professional should fully acknowledge, and express in words, the patient's feelings and concerns—try to see the problem from their point of view.
- **Develop the discrepancy** between what the patient currently does and how they would like to behave in order to fulfil their goals of oral health. In other words**, talk about it.**
- **Roll with resistance** when patient argues against change. This is hard, because there is always the tendency to give the counter arguments, but all this will achieve is the patient pushing hard against you!
- **Support self-efficacy,** which means supporting a patient's confidence to achieve change. Encourage them, express your belief in their ability to change, and acknowledge steps in the right direction. To give an example, when reviewing cleaning when plaque free areas are found, acknowledge these. The patient has done well here.

6.5.3 Some communication hints specific to motivational interviewing

Remember you are engaging with the patient in a dialogue; do not lecture! Four activities, expressed by the acronym OARS are useful:

- **Open-ended questions**: provoke the patient's opinion and do not just ask for a yes/no answer. For example, 'Could you tell me when you brush your teeth?'
- **Affirm the patient**: this means acknowledge and paraphrase what they have just said. For example: 'You have explained you brush every morning, but explained to me you are too tired to do this again at night.'
- **Reflect what the patient is communicating**: this demonstrates an ability to understand from another person's perspective. In the example given you may be in the same boat yourself!
- **Summarize**: for example, 'While you are happy with your morning brushing you do not at the moment think you can do this again at night. Have I got that right?'

6.5.4 Giving information

As explained in the beginning of this chapter is important to provide information, just do not expect this to be all that is involved! In MI the important thing is to provide information with permission to do so, and the patient is willing

and interested to receive it. A three-step process has been suggested in MI for use as a framework for giving advice:

- **Step 1**: elicit the patient's or parent's readiness to receive the information. For example, if a first molar is erupting you might say: 'Look, here is the first grown up tooth coming through. This is hard to clean, may I show you one way of doing this?'
- **Step 2**: provide the information in a neutral fashion first. For example, 'We know these erupting teeth can take over a year to come fully into the mouth; they are hiding below and behind the other teeth and are difficult to reach with a brush.'
- **Step 3**: elicit the person's reaction to the information. In the example given, you might ask to show the difficulty by using disclosing solution, which will give the opportunity to point out the clean occlusal surfaces of the deciduous molars, but highlight the plaque-covered erupting tooth that is below the occlusal plane. At this stage it is quite likely the parent may ask how to solve the problem.

This will take practice. In the cold light of day, with no patient present, these suggestions may seem infantile at best and patronizing at worst. It might be helpful, having read this, to go and listen to others giving dental health advice. You will hear some of the errors suggested in this text and see some of the successes of a different, patient-centred, approach.

6.6 Turning intentions into behaviour

Motivational interviewing is about defining and encouraging the patient's willingness to change. The next key step is to transform the intention into actual behaviour. This is sometimes referred to as *volition* and, again, it concentrates on the client. A definition of volition would be 'use of one's own will in choosing or making a decision'—she did it of her own volition, it was voluntary.

6.6.1 Specify goals and make a plan

Work with the patient to set goals that are realistic, short-term and regularly reviewed. A plan will help the patient remember and feel confident about changing behaviour. For instance, a parent has agreed to brush their child's erupting first molar. Discuss how this might be organized at home.

- **When** will this start and when in their busy day might this be practical? When are teeth brushed and when is it convenient for the parent to take the brush and finish off the erupting tooth? It is obvious that this will be specific to the clients (child and parent).

- **Remembering** can be difficult. Can the parent suggest a mechanism? For instance, a tick chart in the bathroom, a sticker chart might be a possibility with the child adding a sticker each day. If this is a possibility, be prepared to supply the chart and stickers!
- **Write down** what has been agreed, both for the client and in the notes.

6.6.2 Reviewing progress and rectifying problems

Regular reviews of progress are an incentive to change and remind the patient. They also provide the opportunity to identify and correct problems. To give an example, if the goal is to brush an erupting tooth, this should be reviewed soon. It is not sensible to dismiss the family for months; everyone needs to know things are on course next week. Would the family be prepared to come into the surgery, briefly, to check how things are going? If things are not going well on review (in the example, use of disclosing solution shows the tooth is plaque covered), encourage the parent to talk about the problems, listen to them. Then consider the following:

- **Is the task too difficult**, i.e. the parent cannot clean the tooth for some reason? What are the problems?
- **The original plan was impractical and should be reconsidered**. It has being 'forgotten', and finding why this is and whether the client can suggest a different plan is important. Can the health professional work with the family to make it easier for the patient to follow the advice?

6.6.3 Rewards for success

In the chosen example, the reward for success will be a fully erupted, caries-free occlusal surface. Could the practice build in a specific reward for this? Would a 'first molar club', with a specific badge be appropriate in your practice?

6.7 Failure

It would be nice to think that if the approach in this chapter was followed, failures in patient motivation would be rare. This is not the case—failures are common in MI and the art is to lead the patient back to the desired behaviour. Remember, it is their desire that matters, go back to contemplation.

There will be many reasons why patients do not follow advice and many of these are not within your control. With failure, it is worth considering if whether anything could have been done differently and one sensible approach would be to ask the patient! One of us remembers a real surprise when doing this. Oral hygiene of an intelligent professional man was stubbornly dreadful.

He eventually said 'You like the feel of smooth shiny teeth but I prefer them furry'. Another case, that caused laughter from both the patient and the dentist, concerned a patient who loved sweets, but said he thought of the dentist each time he had anything sweet. Asked if he thought of the dentist a lot, he said he did! Reference to this was always the first question at recall, 'Have I been on your mind a lot?'

The importance of the dental team should not be underestimated in this. What the dentist may not be able to achieve, the hygienist, nurse, or dental health educator may or vice versa. Sometimes, it can be something as simple as a different personality taking over that changes the outcome.

Behaviour change in patients can be a slow process. It does not usually happen after one visit. It is often a continuous process of building up trust and rapport with a patient over many visits, continuing to be encouraging and finding small improvements to be positive about. It is also an ethical necessity to be honest with patients, and it may be necessary to confront the patient and tell them they have two new lesions. Ask them what they think about this situation. Notice the patient is drawing the conclusion. Where it is a parent who has failed in care, discussion in the absence of their child is considerate and can be constructive.

It is fortunate in that dentists can recall patients and sometimes, with age and experience, see children of patients now attending the practice. What failed in one generation may succeed in another. However, in the final analysis the teeth belong to the patient.

Dental health educators need to remember the adage, 'you can lead a horse to water, but you can't make it drink'. Ours is the business of creating the environment that makes the horse thirsty. The greatest frustrations may occur where the dentist is powerless to improve things, perhaps because of financial constraints or perhaps because the patient needs help from someone else, which this is not forthcoming. Dental neglect is very distressing and can signal neglect or abuse of an even more serious nature.

Further reading

Gao, X., Chin Man Loe, E., Ching Ching Kot, S., and Chi Wai Chan, K. (2014). Motivational interviewing in improving oral health: a systematic review of randomized controlled trials. *J. Periodontol.*, **85**, 426–437.

Newton, T. (2013). Effective communication with patients to improve motivation. In:Palmer, R. M., Ide, E., and Floyd, P. D. (eds) *A Clinical Guide to Periodontology*, 3rd edn, pp. 33–39. British Dental Association, London.

Ramseier, C. A. and Suvan, J. E. (eds) (2010). *Dental Health Behaviour Change in Dental Practice*. Wiley Blackwell, Oxford.

Ramseier, C. A., Catley, D., Krigel, S., and Bagramian, R. (2008). Motivational Interviewing. In:Linde, J., Lang, N. P., and Karring, T. (eds) *Clinical Periodontology and Implant Dentistry*, 5th edn, pp. 695–704. Wiley-Blackwell, Oxford.

Yevlahova, D. and Satur, J. (2009). Models for individual oral health promotion and their effectiveness: a systematic review. *Aust. Dent. J.*, **54**, 190–197.

Videos

Motivational Interviewing - Good Example - Alan Lyme, YouTube. Available at: https://www.youtube.com/watch?v=67I6g1I7Zao (accessed 4 December 2015).

Dentistry: You Tube videos.

Chapter 7

Caries control for the patients with active lesions

7.1 Introduction

Chapter 4 described caries control measures for everybody, a whole population approach. The emphasis was on oral hygiene, regularly disturbing the biofilm with fluoride toothpaste. The mode of action of fluoride was discussed in some detail to show that this **therapeutic** agent acts topically to interfere with the de- and remineralizing processes and delaying lesion development. The relevance of minimizing sugar intake was discussed. The metabolism of sugar, by micro-organisms in the biofilm, creates the acidic environment for demineralization.

However, what more should be done for those **presenting** with active lesions? This chapter will consider how to find out **why these patients** are developing

lesions. The chapter will then explore further oral hygiene measures that might be useful. It will question how fluoride might be boosted and their diet modified. Specific groups, such as babies and young children, those with erupting teeth, patients undergoing orthodontic treatment, and patients with dry mouths will be individually discussed. Finally, a section will discuss the difficulties of advising carers on helping those who can no longer care for themselves, either though illness, disability, old age, or dementia.

7.2 Explaining an individual's caries experience

The caries activity of any patient, child, or adult, is assessed at the first visit of the patient by noting how many lesions judged as active are present (both cavitated and non-cavitated) and where they are located (see Chapter 3). Please note, this assessment is mainly based on **clinical assessment**. Some companies produce a battery of chairside salivary tests, such as microbiological counts of specific microorganisms, but these are not needed. If the patient is coming for a regular check-up, a history of recent caries activity is available (number of lesions and fillings over the last 1–3 years). This information is most valuable. A yearly increment of one or more lesions detected clinically, would indicate a high rate of lesion formation and progression.

Once a dentist has assessed an individual patient's caries activity as high, an attempt should be made to identify the relevant risk factors **for this patient**. It is possible to interfere with and modify many of these factors, and thus arrest ongoing active lesions, or slow down the disease activity and diminish the rate of progression. Some of the risk factors are listed in Box 7.1. Biofilm control, preferably with fluoride toothpaste as the vehicle of topical fluoride, is essential and diet should be examined and discussed with the patient. Saliva secretion rate may need specific investigation in particular if the patient complains of 'dry mouth' or is on medication.

7.2.1 Biofilm control

If an individual is developing active lesions, aim at enhanced biofilm control. It is essential to focus on this so that the patient understands the role they have to play. As part of explaining the individual's caries experience, consider **why there is a problem** disturbing the biofilm regularly at the sites in question. For instance:

- **Does the patient require help that is not forthcoming?** A baby, infant, child, ill, or disabled person (child or adult), are examples of those who need assistance. If this is part of the problem, consider how to approach this with the carer.

Box 7.1 Factors that may be relevant to the development of active caries

Medical history.

- Medications known to cause a dry mouth.
- Medications containing sugar.
- Radiotherapy for head and neck malignancy (dry mouth).
- Sjögren's syndrome (dry mouth).
- Rheumatoid arthritis (may also have Sjögren's syndrome).
- Diabetes (may be dry mouth).
- Disability.

Dental history.

- Erupting molars.
- History of multiple restorations.
- Frequent replacement of restorations.
- Sudden need for multiple restorations.

Oral hygiene.

- Low frequency of tooth cleaning.
- Toothpaste that does not contain fluoride.
- Paste vigorously rinsed from the mouth.
- Appliance worn, e.g. orthodontic appliance, partial denture.

Diet.

- Frequent sugary drinks and snacks.

Fluoride.

- No fluoride in toothpaste.
- Teeth rarely brushed.

Saliva.

- Low flow rate (unstimulated and stimulated).

Social and demographic factors.

- Poverty.
- Low educational status.
- Unemployed.
- Religion or ethnicity may be relevant.
- In residential care.

- **Perhaps the caries lesion is difficult to access** with a cleaning aid. Suggest how to deal with this. These are the easy ones, provided the patient wants advice and is prepared to take it!

- More difficult are the cases where the patient can remove the biofilm, but chooses not to. This is discussed in Chapter 6.
- **Do not ignore practicalities**. Most basic is a supply of clean water. The money to buy cleaning aids and toothpaste is highly relevant.

It might be sensible to list the difficulties (as you see them and later, after discussion, as the patient sees them) in the notes. Make sure that the patient fully understands that he/she alone is responsible for success or failure. The patient should not be allowed to transfer the responsibility for solving the problem to you. It is not you that controls caries, it is the patient. So they lie in the dental chair, 'here is my mouth, treat me', but you cannot do this. 'Yes, here is your mouth, let me show you how to look after it'.

7.2.2 Investigating diet

Questions about diet are obligatory when the patient presents with active caries lesions or a history of multiple restorations that are frequently replaced. Dentist and patient (or parent where a child is involved) are searching for an inappropriate dietary habit that may partly explain the caries incidence.

Some groups may have a particular problem with caries because of dietary factors. It cannot be said the particular group **will** have a problem because caries is a multifactorial disease and the relationship will not be simple. However, the groups are worth listing because they should sound 'warning bells':

- Infants and toddlers provided with a dummy containing a sugar solution, or sugar-containing feeding bottle used at bedtime, or this sweet liquid suspended in the cot for use during the night.
- Children, adolescents, and young adults with a daily consumption of snacks, biscuits, or soft drinks and fruit juices, which may, or may not, have added sugar.
- Athletes or joggers taking sugar-containing sport supplement drinks.
- Workers subject to occupational hazards, such as food sampling, those working in the confectionary and bakery industry, and those on a monotonous job, such as a night shift.
- People with an increased frequency of eating because of a medical problem, e.g. gastrointestinal disease, eating disorders, uncontrolled diabetes.
- People of any age on long-term and/or multiple medications. Are these sugars based and/or do they cause a dry mouth?
- Those with an increased carbohydrate intake due to a medical problem, e.g. Crone's disease, chronic renal failure, other chronic illnesses, malnutrition, or failure to thrive.
- Those with reduced salivary secretion leading to a prolonged clearance rate of sugars and a Stephan curve that takes longer than usual to return to a neutral

Box 7.2 Causes of dry mouth

Medications—consult a formulary.

- Antidepressants.
- Antipsychotics.
- Tranquillizers.
- Antihistamines.
- Diuretics.
- Anti-Parkinsonian drugs.
- Appetite suppressants.
- Check liquid medicines, pastilles, and inhalers for sugar base.

Diseases and disorders.

- Sjögren's syndrome.
- Rheumatoid arthritis.
- Lupus erythematosus.
- HIV/AIDS.
- Alzheimer's disease.
- Parkinson's disease.
- Strokes.
- Cystic fibrosis.
- Dehydration.
- Drug abusers.

Radiotherapy in head and neck region.
Chemotherapy.

pH, e.g. those on medications causing reduced salivary flow (see Box 7.2), Sjögren's syndrome, irradiation in the head and neck region.

- Drug abusers who have a craving for sugar and a prolonged clearance rate, as a result of reduced salivary secretion.
- Elderly people with a tea and coffee routine where cakes and biscuits are consumed in-between meals. How many spoons of sugar are added to each cup?

The diet of patients presenting with multiple lesions should be examined to try to determine whether any changes should be advised as part of a caries control programme. This is called diet analysis and there are two principal techniques for determining food intake. One is to record the dietary intake during the preceding 24 hours, the so-called **24-hour recall** system. This involves careful history taking, and relies on the patient's memory and honesty. The other

method is to obtain a **3–4-day written record,** with the patient recording food and liquid intake as it is consumed. This relies on the patient's full co-operation, as well as honesty.

Both forms of diet recording suffer from the disadvantage that the record may not be representative of the diet consumed over a much longer period of time, although it is this which is likely to have been responsible for the caries status with which the patient presents. Thus, a diet history is an unscientific tool and must be interpreted with caution. However, one of its strengths is the patient involvement, particularly in the written record. It is really the patient who is taking the lead in the diagnosis and this may be of enormous assistance in behaviour change if they wish it.

The diet sheet in Figure 7.1 can be used. First explain the reason for the investigation to gain the patient's consent, explaining that their help is needed to find the cause of their dental decay. The cause is likely to be related to what they eat and drink, and for this reason it is necessary for them to record everything eaten and drunk over a 4-day period, together with the time;

Diet Analysis

	THURSDAY		FRIDAY		SATURDAY		SUNDAY	
	Time	*Item*	*Time*	*Item*	*Time*	*Item*	*Time*	*Item*
BEFORE BREAKFAST								
Breakfast								
MORNING								
Mid-day meal								
AFTERNOON								
Evening meal								
EVENING and NIGHT								

Figure 7.1 Form on which a patient may record diet over a 4-day period. This has been designed to highlight the between-meal snack, thus facilitating discussion with the patient when the sheet is returned. On the reverse side of the sheet there are simple instructions explaining how it should be filled in.

in addition any medication should be entered. They are requested to keep the diet sheet with them and fill it in at the time to avoid missing anything. Quantities of food consumed are not specifically requested, but it should be stressed that nothing should be changed because a record is being kept. Health professional and patient are partners in the investigation, and the object of the exercise is to help, not condemn.

Although this strategy is useful for many patients, it may be inappropriate for others. Sometimes variable dietary habits will mean that the 4-day record is inappropriate or even misleading. Shift workers will have different dietary habits from week to week, as may those whose work necessitates frequent trips abroad. 'Bingers' will be unwilling to commit their dietary pattern to paper. A medical history may reveal unstable health conditions such as intermittent ulcer problems, and thus a 4-day record may be inappropriate. However, if the patient understands the purpose of the record, they may suggest how best it is kept. For instance, a shift worker may record 2 days on duty and

DIET ANALYSIS (See notes on other side) * = 2 spoons of sugar.

	THURSDAY		FRIDAY		SATURDAY		SUNDAY	
	Time	Item	Time	Item	Time	Item	Time	Item
BEFORE BREAKFAST	7·45	Tea *	7·00	Tea *	7·00	Tea *		
Breakfast	9·00	Coffee *	8·45	Coffee *	10·00	Tea * 2 pieces of toast		
MORNING	10·00 10·45	Coffee * Rolland butter Coffee *	9·30 10·45 11·45	Coffee * Roll and butter Coffee * Coffee *	11·00 12·00	Coffee * Coffee *	10·45 11·30	Tea * Tea *
Mid-day Meal	12·30 1·30	Coffee * Coffee *	1·45	Cheese & onion sandwich ½ lager	1·0	Coffee * 1 muesli biscuit	2·0	Beef: roast potatoes carrots: greens fresh pear Tea *
AFTERNOON	2·30 3·45 4·15	Coffee * Coffee * Coffee *	2·30 3·15 4·00 5·00	Coffee * Coffee * Coffee * Coffee *	3·00 4·15	Tea * Tea *	3·30	Tea *
Evening Meal	7·30	Country hash Tea *	9·00	Lasagne Tea *	7·00	Spare Ribs, Rice Tea *	5·00 6·30	Tea * Coffee *
EVENING & NIGHT	9·00 10·30	2 muesli biscuits Tea * Pear	10·00 11·15	Tea * Tea * 2 biscuits	9·30 11·00	Tea * Tea *	8·00 10·00	Coffee * Cheese & biscuit Tea *

Figure 7.2 Diet sheet completed by a middle-aged secretary with a very high incidence of caries. This lady returned to the surgery saying that she now realized that drinking sweetened cups of tea and coffee was the probable cause of her caries.

2 days off. Those with a medical history may record typical days when they are 'well' and other days when they are 'ill'. Finally, it must be appreciated that a patient may not always tell the truth, although if they know **what** to lie about, progress has been made!

Figures 7.2 and 7.3 are examples of two completed diet sheets and possible management strategies will be discussed in Section 7.5, 'Dietary advice following diet analysis'. For now, notice the sheets show a high and frequent sugar intake. This is inevitably relevant to the caries status with which these patients presented. Can/should anything be done to modify this? Be aware that parents of smaller children may often **not** inform you correctly because they know that they themselves have stimulated bad habits with sugar intake. Children in the teenage group simply behave as this group does— they can't care and will not tell.

DIET ANALYSIS

	THURSDAY	
	Time	10 attacks Item 1 pre-bed
BEFORE BREAK-FAST	7.15	1 pint skimmed milk milk shake
Breakfast	8.30	Black coffee + Sugar
MORNING	10.00 12.00	White coffee + Sugar 2 cheese rolls Bag Crisps, Cake Twix White coffee + Sugar
Mid-day Meal	13.30	2 cheese rolls Cake 1 mint Apple
AFTER-NOON	15.30 17.00	White coffee + Sugar Tea + Sugar 4 Biscuits
Evening Meal	19.30	Pizza Ribena Ice Cream
EVENING	21.30 22.30	Glass of coke Glass of coke

Figure 7.3 One day in the initial diet sheet of a 24-year-old mechanical engineer

Box 7.3 Some clinical signs of oral dryness

- Mirror sticks to mucosa or tongue.
- Saliva frothy.
- No saliva pooling in the floor of the mouth.
- Tongue papillae shortened, tongue lobulated, or fissured.
- Gingivae smooth.
- Mucosa looks glassy.
- Debris on palate or sticking to teeth.
- Caries lesions.

7.2.3 Investigating saliva

Saliva is a protective fluid as far as the mouth is concerned. A low secretion rate leads to reduced elimination of microorganisms and food remnants, impaired neutralization of acids, and a reduced ability to repair minor demineralizations (see Chapter 2). Increased caries activity is often seen in persons with a reduced rate of salivary secretion. However, some patients are aware of dry mouth (this is called **xerostomia**). Others with reduced salivary flow (**hyposalivation**) do not realize that they have a dry mouth. Dentists can sometimes detect a lack of saliva during the course of a clinical examination. Box 7.3 lists some clinical features of oral dryness.

Unstimulated and stimulated salivary flow rates should be measured where a dry mouth is suspected, or in cases where a high caries incidence cannot be explained.

Normal values are:

- Unstimulated: 0.3–0.5 ml/min.
- Stimulated: 1–2 ml/min

Before measuring salivary flow, the patient should not eat or drink for 90 minutes. Saliva should initially be cleared from the mouth by rinsing with water. For consistency of measurement, repeat collections should be taken at the same time of day.

Unstimulated salivary secretion rate

The patient sits quietly in the dental chair for 10 minutes, without chewing or swallowing, but spitting into a disposable cup. The volume of saliva per minute is measured by aspirating the saliva into a disposable graduated syringe. The secretion rate is then expressed in millilitres per minute. When the secretion

rate is very low, the saliva collected may be frothy and difficult to measure. In such cases the addition of a measured amount of water will eliminate the froth and so facilitate measurement.

Stimulated salivary secretion

The patient is asked to chew on a piece of paraffin wax. Saliva formed over 5 minutes is expectorated into a disposable cup. The volume of saliva secreted is measured by aspirating the saliva into a graduated syringe.

Causes of dry mouth

If the mouth is dry, consider why this is. Box 7.2 lists common causes of dry mouth and it should be noted that many of these may be permanent. Thus, the patient is at high risk of caries progression; it may not be possible to alleviate the cause and they will remain at high risk.

Hundreds of **medications** have a side effect of salivary gland hypofunction and this will be listed as a complication in a formulary of medications. Thus, you do not have to rely on memory alone—look it up in a formulary and the instruction sheet that comes with the medicine, which will list the contents of the medicine and the side effects. Always check any liquid medication is not in a sugar syrup form. Pastilles that have to be sucked should also be checked to be sure they are not sugar-based. An example would be nystatin pastilles prescribed for oral fungal infections, often caused by a dry mouth. Fortunately, most liquid medicines and pastilles are now sweetened with a non-cariogenic sugar substitute. In addition, check the constituents of any inhaler used for asthma. Some contain lactose in the propellant.

Many **systemic diseases** cause dry mouth. Sjögren's syndrome is an autoimmune disease of the salivary glands, where the glands are destroyed by a lymphocytic infiltration. It may be accompanied by dry eyes because lacrimal glands are also affected. There may also be another connective tissue disease, such as rheumatoid arthritis or lupus erythematosus. Patients with HIV/AIDS may also have a lymphocytic destruction of salivary glands as a consequence of medication. Those with attention deficit hyperactivity disorder (ADHD) may also have a dry mouth as a result of their medication.

Diabetes, particularly when poorly controlled, can lead to altered salivary secretion. Saliva may also be short in Alzheimer's disease, Parkinson's disease, strokes, cystic fibrosis and dehydration. Once again, do not rely on your memory here. Have available a suitable reference, such as *Scully's Medical Problems in Dentistry*, where it is possible to look up any disease and read the dental relevance. Get into the habit of looking up a patient's diseases after taking the medical history.

Drug abusers frequently have dry mouth as a consequence of their opiate addiction. To make matters worse, they have a chaotic lifestyle and oral hygiene may be

very poor. They also crave sugar as a result of the drugs they use. Methadone may be used to wean patients off addictive drugs, but sometimes this is sugar-based.

Radiation therapy, used in head and neck malignancy may also damage the salivary glands and result in dry mouth. **Chemotherapy** may also disturb the salivary secretion, although this is normally temporary.

7.3 Oral hygiene advice for those with active lesions

What more help with oral hygiene might be required for those with active lesions? If possible, show the patient the lesion and it is often possible to relate it directly to the biofilm (Figure 7.4), which makes the oral hygiene advice particularly pertinent. What brush is the patient using? Is it possible to help them use it better or might an electric toothbrush help?

Where an enamel lesion is present approximally, it is usually possible to show the patient the lesion on the bitewing radiograph, then show them where it is in the mouth before asking if they would be prepared to floss this area, daily or perhaps twice a week. Floss is difficult to use and it is worth spending time demonstrating

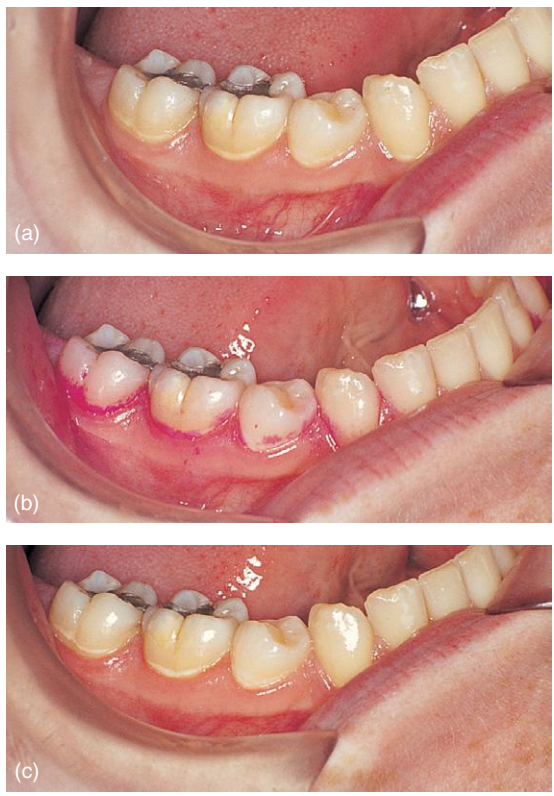

Figure 7.4 Caries of the enamel at the cervical margin of lower molars: (a) White spot lesions covered with plaque. (b) Red dye has been used to stain the biofilm so the patient can see it clearly. (c) The patient has now removed the stained biofilm with a toothbrush: the white spot lesions are now very obvious. Note: they have formed in an area of biofilm stagnation and this can be shown to the patient to demonstrate the importance of brushing.

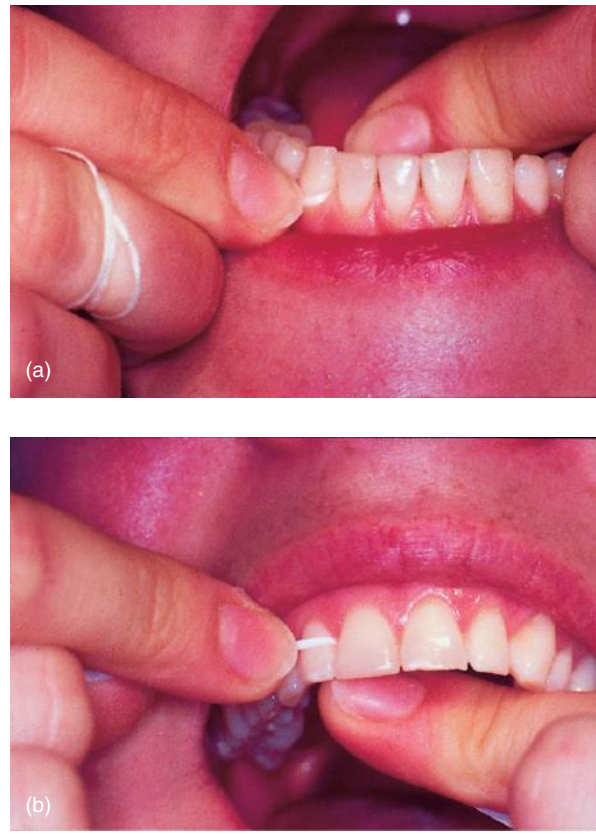

Figure 7.5 (a) Use of dental tape for interproximal cleaning of the lower teeth. Two index fingers are used to control the floss. (b) Use of dental tape for interproximal cleaning of an upper contact. Note how the controlling fingers are close together and the tape is wrapped around the surface of the tooth being cleaned.

the technique (Figure 7.5). A floss holder can help in these circumstances (Fig 7.6). Where root caries lesions are present, an appropriate size of interdental brush should be selected and the patient shown how to use it (Fig 7.7). Each of these cleaning methods should be checked at an appropriate recall.

Figure 7.6 Floss holder.

Figure 7.7 Patient using interdental brush to clean an approximal root lesion developing cervical to an amalgam restoration. Note this patient has active periodontal disease, you can see pus buccal to the premolars.

7.3.1 Professional plaque control

In caries active patients who, for some reason, do not master plaque control themselves and/or in patients with decreased salivary secretion, additional plaque control in the form of professional tooth cleaning can give the patient some extra support for a short time.

The clinical procedure is as follows:

- Disclose plaque.
- Remove plaque with a low abrasive, fluoride-containing polishing paste. A handpiece (rotating up to 5000 rpm) is used with a pointed bristle brush for the fissures and a rubber cup for free, smooth surfaces. For proximal surfaces the paste is applied with an interdental brush. Floss all contact points to ensure the biofilm is disturbed.
- Disclose again to check as much plaque has been removed as possible.
- Apply either a 2% NaF solution or fluoride varnish to sites with active lesions. The solution is applied to the dried lesions with a cotton pellet, and the varnish with brush or sponge applicator. The varnish has a high fluoride concentration (typically 22 mg/ml, 2.2% sodium fluoride, 22,600 ppm fluoride) and it is contraindicated in children under 3 years who may swallow some of the product.
- Avoid eating, drinking, or rinsing for 30 minutes after application. Toothbrushing should recommence on the day following application.

This is a highly expensive treatment programme, but it may be justified for a limited period in the management of some highly caries active patients. Recall intervals of 1 month can be used.

7.3.2 Opening cavities for cleaning

Cavitated deciduous teeth can be opened by the dentist to allow access for the toothbrush. The technique is often called **non-restorative cavity treatment (NRCT)** and the aim is to put the management of caries where it has to be, with the parent. This management can be chosen in preference to restorations in this temporary dentition (see Chapter 5.4.1). The aim is to arrest the lesion by brushing and fluoride treatment, and modify behaviour so that when permanent teeth erupt, new habits have been established.

7.4 Fluoride

There are three main ways where the effect of fluoride can be boosted in those developing several active lesions—use of high concentration toothpastes, mouthwashes, and varnish application. Please note, *Cochrane reviews* show no clear evidence that any single modality (toothpaste, mouthwashes, or varnishes) is more effective than any other, but there may be an additive effect when mouthwash/varnish is used in addition to fluoride toothpaste.

- **Prescription of high-fluoride concentration toothpaste**. A paste with fluoride concentration of 2800 ppm fluoride may occasionally be useful in patients who are not able to care for themselves, and present with multiple lesions, poor oral hygiene, and a highly cariogenic diet. However, the dentist should avoid using it for more than 1–2 months because dental caries is **not** caused by too little fluoride—or fluoride deficiency. Similarly, a paste with 5000 ppm fluoride may be prescribed for those with dry mouths and in functionally-dependent adults (see Sections 7.6.4, 'Patients with dry mouths', and 7.6.5 'Functionally dependant adults'). Remember fluoride can never solve the caries problem unless biofilm removal and diet are improved. None of these products should be freely available in the bathroom of families with children, because if ingested by small children they would cause fluorosis
- **Fluoride mouthwash**. These can be prescribed for patients 8 years and above for daily use in addition to toothpaste containing at least 1350ppm fluoride. The fluoride concentration is 0.05%. A parent should supervise and the patient should rinse for 1 minute and then spit out. Rinses should be used at a different time to toothbrushing to maximize the topical effect, which relates to frequency of availability. Note, fluoride rinsing should not immediately follow toothbrushing, as it may wash away the toothpaste. (The fluoride concentration of the toothpaste is 1000–1500 ppm, while the concentration in the rinse is 225 ppm, so it would be stupid to wash the high fluoride paste away by vigorous rinsing.)

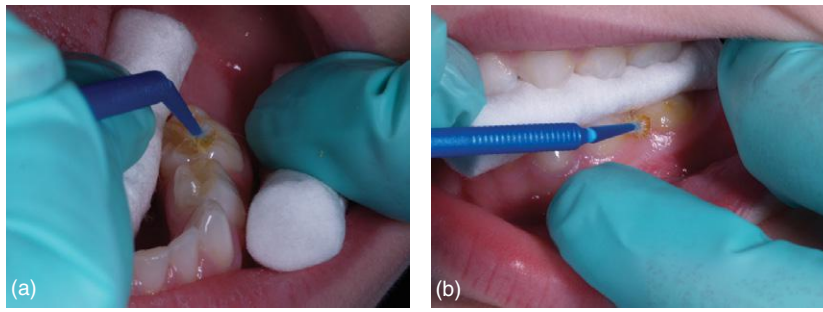

Figure 7.8 Varnish application to the occlusal surface (a) and proximal surface (b). Note the cotton wool isolation that was placed prior to charting.

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- **Fluoride varnish**. Varnish application can further influence the arrest of developing lesions on enamel and root surfaces. Figure 7.8 shows varnish application immediately after examination of teeth for caries lesions. The teeth are already isolated, clean and dry. Using a small brush to apply the varnish to pits, fissures and approximal surfaces, takes only seconds. The fluoride concentration in varnish (22,600 ppm fluoride) is high and great care should be taken to ensure only a small quantity of varnish is applied to teeth particularly in young children. This application deposits a fluoride reservoir into partly dissolved enamel crystals. The professional does the application and apart from attending the appointment, no further compliance is needed from the patient. However, the cost-effectiveness is questionable because it has to be repeated two or three times per year.

7.5 Dietary advice following diet analysis

Although the link between sugar and caries is irrefutable, there is no evidence that dietary advice at the individual level is effective. So is analysis and advice a waste of time? Chapter 4 refers to the obesity epidemic and the responsibility of all health professionals to try to encourage the populations to reduce sugar intake. Look again at the two diet sheets Figures 7.2 and 7.3. It will be obvious to that in these two cases the frequency and amount of sugar consumed is of relevance to the clinical presentations of active lesions. Can we, by advice, change this? For a start, has the wrong question been asked! It is the **patient** who will decide if they will change anything, but it is suggested they would have every reason to be angry if they had not even been given the opportunity to consider this.

As will be illustrated, a possible approach is saying what actually happened in these cases. The lady who kept the diet sheet in Figure 7.2 positively bounced into the surgery waving the sheet in triumph! She realized the sugar in tea and coffee was the problem, and had changed this and lost weight already, and was delighted by this. There were things to discuss, such as how little food she had apart from the sweetened drink, the lack of fruit, and often no breakfast or lunch.

A very similar sheet was returned by the lady whose bitewing radiograph shown in Figure 5.5a. She worked as an executive secretary with sweetened coffee on her desk and was always sipping this. She was an example of baby bottle caries in an adult! However, she was a dentist's daughter whose father had told her he could cope with anything she developed in the way of caries with the dental drill. The current dentist explained what she thought was the relevance of the continual sweet drink and then listened to the patient. The patient's attitude of relying on the dentist to fix her teeth appeared unchanged and she cancelled her subsequent appointment with a polite letter saying she thought she was coming to be 'treated'—by this she meant more restorative dentistry. You cannot win them all and in the final analysis the teeth belong to the patient!

However, make sure that your advice is clearly stated in the records. Occasionally, this has a surprising effect especially if the patient is asked to sign a statement in the records. They then appreciate that this is not just a routine response but a serious comment from a responsible dentist.

So how, practically, does one go about analysing and discussing the record the patient has carefully and at some inconvenience, produced? Here is one approach:

- Use a **highlighter pen to mark** items containing sugar. Encourage the patient to identify these and it will become apparent if the patient realizes which items are sweet.
- Mention again the **relevance of sugar** and the role of the biofilm.
- Count the **number of sugar attacks** and write the number at the top of each day. This gives the opportunity to explain the relevance of frequency of sugar attack. The amount of time that plaque—the dental biofilm—remains acid and capable of causing demineralization, varies depending on such factors as the consistency of the food, salivary flow, and salivary clearance rates. These scientific deliberations will be lost on the patient and they need a simple (but it is scientifically simplistic) message.
- The **message** suggested is that after a sugar attack the plaque will, as a rule of thumb, remain acid for an hour and thus eight sugar attacks will equal 8 hours of attack. It is known from examination of the mouth that this is too long. Anything sweet close to falling asleep is especially damaging as salivary flow is reduced or totally arrested at night, so the plaque may stay acid all night.

- Ask the **patient to suggest** if any changes could be made. Would it be possible to restrict sugar to mealtimes? Could the number of sugar attacks be reduced to 2-3 per day related to the meals?
- Note to **adequacy of main meals**. If sugar is reduced will the patient be hungry and crave a sweet snack? (Figure 7.2).
- **Record** any changes agreed in the notes and enquire about how things are going at the next appointment.

Figure 7.3 is one day in the diet sheet of a 24-year-old mechanical engineer who made things very easy for the dentist by commenting that he tended to graze, rather than eat conventionally. His grazing's were frequently sweet and, although his oral hygiene was quite good, his caries status showed that something needed to change. White spot lesions were developing around the margins of restorations and the dentist's bitewing radiographs, taken with a year's interval, indicated that approximal enamel lesions were probably progressing into dentine. The diet sheet shows 10 separate sugar attacks/day, including a sugary drink just before bed. All drinks were cariogenic and the main meals were inadequate, because he ate while he worked. This has relevance because cutting out sugar may mean this man is really hungry and the temptation to resume the sweet snack may be irresistible.

Here are the **suggestions** for dietary change that, after discussion, the patient thought were 'worth a try':

SUGGESTIONS

1. Aim 2–3 sugar attacks/day
2. Never sugar before bed
3. Coffee and tea—try no sugar
4. If coke; use diet variety
5. Water is safe (patient hates milk!)
6. Try savory roll to eat at work
7. Try to eat more lunch and reduce 'grazing'!
8. Eat lots in evening.
9. Beer is not cariogenic!

Figure 7.9 is one day from the second diet sheet produced by this patient. He had made huge changes. Whether he stuck to these is unknown, but over subsequent years his caries problem was controlled. This highly motivated young man is an example to all! On the other side of the coin was the mature teacher with a similar high sugar, grazing habit. A similar approach was taken and, on recall, he commented he thought about the dentist every time he had sugar. When the dentist asked with a smile if he thought of the dentist a lot he replied, with a twinkle, that he did! This story is amusing, but the interesting side to it

DIET ANALYSIS

	THURSDAY	
	Time	~~Attacks~~ Item
BEFORE BREAK-FAST	7.30	White coffee
Breakfast		
MORNING	10.00	2 cheese + salad rolls Crisps Cake White coffee
Mid-day Meal	13.00	2 cheese + salad rolls Apple
AFTER-NOON	15.30 17.00	Glass of Orange Cup of Tea
Evening Meal	18.00	Pizza Garlic Bread Salad
EVENING	21.00 23.00	Diet coke Beer

Figure 7.9 Second diet sheet produced by the 24-year-old mechanical engineer. Figure 7.3 shows the original diet sheet. The text lists possible changes that the patient thought might be worth a try.

is that the patient had the message and he might wish to use it one day, perhaps when a dentist tells him he needs several new fillings.

Thus far, patients who are in charge of what they eat and drink have been considered. Often, however, a patient is not in charge of the shopping and will simply attack what is in the fridge. It can be important to have the person who stocks that fridge in the consultation appointment. Have a look at Figure 7.10. This shows rampant caries in a young man brought (one might say 'dragged'!) along by a desperate fiancée planning a wedding and anxious that the photographs should not show the dental devastation. Oral hygiene was very poor, and all this man's drinks were fizzy and sweet. Oral hygiene improved and the fiancée, who stocked the fridge, stopped buying sweet drinks; restorative dentistry, simple adhesive restorations, restored appearance, and the wedding photographs were a triumph. In addition, there was a bonus in this story. It turned out the fiancée was an assistant and carer in a residential home, and said all she had learnt would be of great help in her work. This couple allowed the dentist to show her how to brush the teeth of someone who could no longer do this for themselves. She brushed her husband's teeth and he pretended he needed the help.

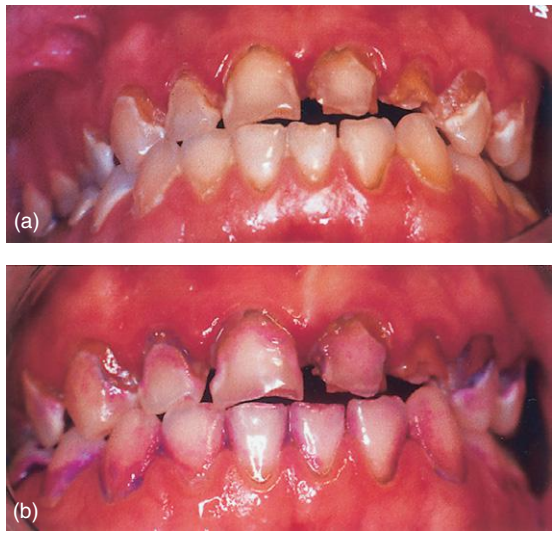


Figure 7.10 (a) Rampant caries in a young man. Poor oral hygiene is combined with a high sugar diet. (b) Disclosing shows the heavy biofilm deposits.

7.5.1 Safe snacks and drinks

It is very difficult to draw up a list of snacks that are safe in all respects. To give examples, cheese is safe for teeth, but its high content of saturated fat, and often salt, will not please cardiologists. Fruit is less cariogenic than sweets, but most fruit juices are very high in sugar and cannot be regarded as a safe drink—dried fruits, such as raisons and apricots, have high sugar content and cannot be considered as safe snacks. Many fruits are acidic and they, and their juices, may cause acid erosion if eaten excessively. Nuts are a safe snack for older children and adults, but should not be salt coated—bread often contains a little sugar, but is better than a sweet cake or biscuit.

The safe drinks for teeth are water and milk. Should diet drinks, sweetened with sugar substitutes, such as aspartame, be recommended? Probably not, because they are erosive and it is best not to develop a sweet taste, so it would be better to give children plain water. This said, patients who take sweetened tea and coffee or cacao-beverages may dislike the drink without sugar, but the sugar substitute (see Chapter 2) can be very useful.

7.5.2 Dietary misconceptions

It is time to lay a few ghosts! One serious misconception is that only refined carbohydrates (sucrose or white sugar) are harmful to teeth, while other carbohydrates are not. Sucrose is regarded as the 'arch criminal' because it is the most abundant sugar. It is used by food manufacturers all over the world as a

food ingredient and it is readily metabolized by bacteria. However, other sugars, such as glucose, fructose, dextrose, glucose syrup, honey, corn syrup, invert sugar syrup, molasses, treacle, and maltose are also bad for teeth. In addition brown sugar is just as bad as white. See Table 4.2 in Chapter 4 for the many alternative names for sugar your patients may find on food labels.

Health foods are fashionable. It has been suggested that fibrous foods, such as apples and carrots 'clean' teeth, removing plaque. While fibrous foods are preferable to a sucrose snack, there is no evidence that fibrous foods clean teeth. Another popular health food is honey, but this so-called 'natural' sugar is just as cariogenic as other sugars. Many brands of muesli contain both sugar and honey. Naturally-occurring sugar in fruit juices makes these products just as cariogenic as fruit squashes.

Finally, it is very common for patients who are asked to give up sugar in tea and coffee to reduce the amount of sugar (say one teaspoon instead of two), rather than giving it up completely. Thus the frequency of pH fall may not be altered. It is important to check that patients really understand the message; otherwise they may make considerable effort to no avail. Please look back to Chapter 4.4.1, which lists some questions you may be asked about sugar.

7.6 Special groups

There now follows advice specific to certain groups to act as checklists for dental health professionals.

7.6.1 Babies and young children

Some children between 12 and 30 months have a special caries pattern that differs from older children. Lesions are found on buccal and approximal surfaces of maxillary incisors and first deciduous molars this pattern reflecting the particular environmental conditions created if infants are frequently fed from a bottle or dummy containing a sugary solution (Figure 7.11). The upper incisors are the most 'vulnerable', whereas the lower incisors are somewhat protected by the tongue and the copious saliva secreted around them from the submandibular and submaxillary glands. Rampant caries may develop and this is sometimes called bottle caries, although early childhood caries (ECC) is the term more commonly used.

The teeth may break down and lesions progress rapidly. Lesions are extensive and frequently affect free smooth surfaces. Parents may associate this with developmental defects ('they came through like that'), rather than a combination of inadequate or no cleaning, and use of a bottle on demand. Sometimes the bottle of sweet solution is hung on the side of the cot so that the baby can suckle throughout the night.

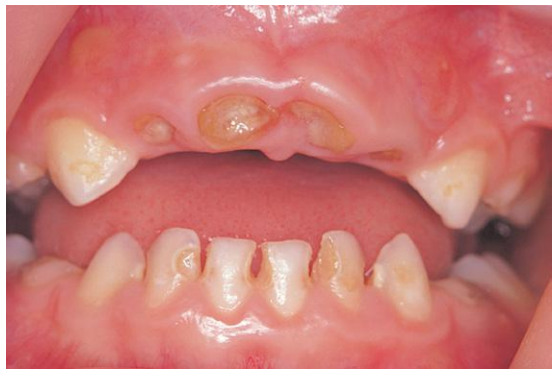


Figure 7.11 Rampant caries of deciduous teeth. This child continually sucked a dummy that was filled with rosehip syrup.

The salient point here is that this group cannot look after themselves. They rely on carers, usually parents, for toothbrushing and choice of diet. The dental team need to see the child with the relevant carer, **as soon as the teeth erupt**. It is not acceptable to wait until cavities form. The following advice is needed:

- As soon as teeth erupt, parents/carers should brush them with a smear of toothpaste containing not less than 1000 ppm F last thing at night and at one other time during the day. **Toothbrushing is mandatory, not negotiable**! See Chapter 4 (Section 4.2.1, 'Toothbrushing for babies and toddlers') for advice on brushing teeth of babies and toddlers.
- Breastfeeding provides the best nutrition for babies.
- From 6 months of age, infants should be introduced to drinking from a free flow cup and from the age of 1 year feeding from a bottle should be discouraged.
- Plain water and milk (unsweetened) are safe for teeth. Fruit juices and sugar-containing drinks should be avoided.
- Sugar should never be added to weaning foods or drinks.
- A dummy or bottle containing a sugar solution should never be used.
- The frequency and amount of sugary food should be kept to a minimum.
- Sugar-free medicines should be recommended.
- On finding early childhood caries, be sensitive and sympathetic to the fact the parents may feel guilty, and may deny it is anything to do with them.

It is also really difficult to break a sweet bottle habit. What advice can be given? Obviously, this must be discussed with the parent and there are two possibilities. The first is to dilute the bottle with water, gradually increasing the water and decreasing the sugar content. Try to achieve only water in the bottle within a week. The alternative is for the baby to go 'cold turkey', which means giving up the sweet bottle and having only water at once. This may be preferable if the baby already has a habit of drinking only water sometimes, but there

will be a screaming child for days and nights. Whichever method is chosen, it is going to be hard for the parents to achieve and they need plenty of support from you. Many of these parents are socially deprived, will give up very easily, and will say that their child is in need of sugar!

7.6.2 Erupting molars

First permanent molars erupt around the age of 6 and second molars around 12 years. Eruption of a tooth may take from 1 to 3 years, and during eruption the gingivae are red, swollen and easily bleed because the bacterial load in such regions are not sufficiently controlled. This bleeding is why parents may avoid cleaning the area for a while and this enhances massive accumulations of bacteria around the erupting tooth, including on the occlusal surfaces. The occlusal surface is easy to miss with the brush because it is below the line of the arch and often brushes are too big to be able to clean properly. It is recommended to use a 'solo' brush (Figure 7.12a). Parents should be shown how to specifically access this surface for cleaning with the brush coming in at right angles to the arch (Figure 7.12b). When mothers visit the clinic explain the problem and 'see them in action' brushing the erupting tooth. Recall child and parent to the practice to check this brushing during eruption. Once this is being done satisfactorily, recall periods can be extended. Remember, fissure sealants cannot replace proper removal of dental biofilm, but may be a feasible adjunct to tide a child over where brushing is still poor despite advice. However, this is seldom needed if one is able to teach parents to clean the teeth properly!

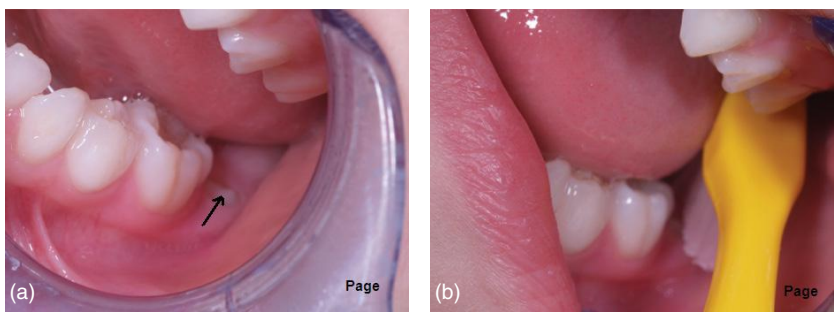
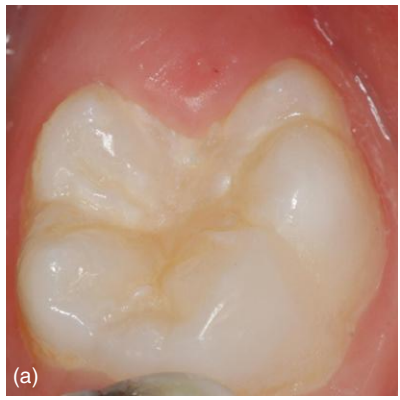
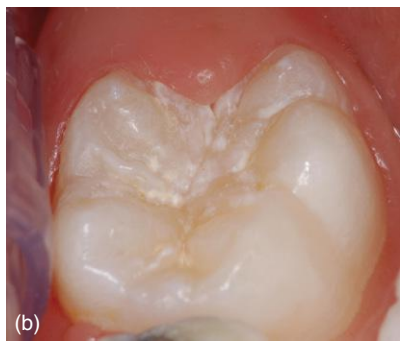


Figure 7.12 (a) The occlusal surface of this first permanent molar is at its most vulnerable during the period from first erupting until it is in occlusion (arrow points to erupting tooth). (b) Brushing the occlusal surface of the erupting molar. The brush is brought in at right angles to the arch.





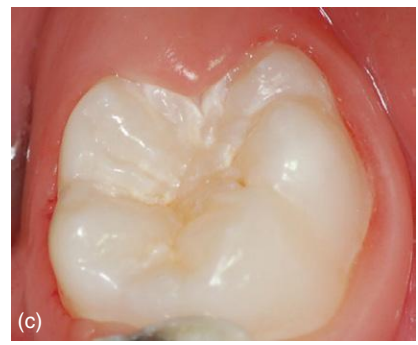


Figure 7.13 (a) The occlusal surface of this erupting first molar is covered with plaque. (b) Plaque has now been removed and the tooth surface dried. Notice the active white spot lesions and the treatment needed is toothbrushing instruction and training in home-based plaque control with fluoride toothpaste. At this visit topical fluoride varnish is applied. (c) Three months later the lesion is inactive. There is still a white spot close to the gingival flap, but this lesion is inactive with a smooth and shiny surface.

Courtesy of Joana Carvalho.

In Figure 7.13 notice the extreme importance of cleaning and drying the occlusal surface to see what is happening. The lesion is not visible (Figure 7.13a) until the plaque is removed with a brush and the tooth is dried. Only now can the white spot lesion be seen (Figure 7.13b) and shown to the parent when giving help with cleaning. After 3 months of brushing by the parent, the lesion is inactive (Figure 7.13c). In the area close to the distal gingival flap, a white spot is still visible, but its surface is shiny and smooth, indicating inactivity. This tooth does not need fissure sealing.

7.6.3 Orthodontics

Orthodontic appliances create multiple stagnation areas and the likelihood of visible lesions developing is high. To control this:

- Treatment should not be started unless oral hygiene is good and gingivae are not inflamed, which indicates good plaque control. How often do orthodontists respect this advice?

Figure 7.14 A patient is shown how to clean around an orthodontic appliance. On the right hand side of the figure you can see a bracket, an elastic, and the arch wire. A small toothbrush has been chosen and the patient is shown how to place the brush so it straddles the appliance and touches the gums. Note there is blood on the brush. This indicates inflamed gums and it is very important to explain the relevance of this to the patient.

- Orthodontic patients should see a hygienist after the appliance is fitted to check cleaning (Figure 7.14). Recall should be frequent, particularly if cleaning is poor.
- A fluoride mouthwash for daily use may be suggested to be used at a different time to toothbrushing. Remember the fluoride does not protect the teeth if cleaning is poor. At best, it influences the rate of lesion development. Choose a bland-tasting, alcohol-free, mouthwash that the child does not find astringent.

The devastation caused by an orthodontic appliance, with poor oral hygiene and a high sugar diet is seen in Figures 7.15 and 7.16. The appliance has been removed in Figure 7.16 and cleaning has improved. The white spot lesions are now arrested (shiny and hard), but the damage has been severe, the appearance is very poor and the lesions will remain as scars. Such teeth are not more at risk to further caries attacks once they are kept clean.

7.6.4 Patients with dry mouths

A dry mouth is very unpleasant. The causes of dry mouth are listed in Box 7.2 and the oral consequences in terms of high rate of dental caries development have been emphasized earlier. The following advice (general and dental) will be helpful:

- Sip water frequently all day long to moisten the mouth. Avoid using sugar-containing drinks or boiled sweets for this purpose, because sugar should be kept to a minimum to control caries.

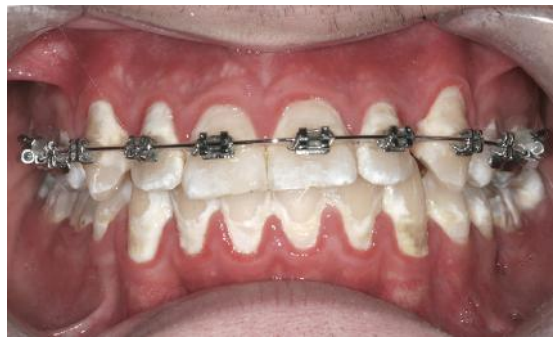


Figure 7.15 Here is a disastrous situation! Oral hygiene is very poor, the gums are inflamed, and numerous active white spot lesions are obvious.

Reproduced from *Dental Update* (ISSN 0305-5000), 42, Dowsing, P., et al., 'Emergencies in orthodontics Part 1: management of general orthodontic problems as well as common problems with fixed appliances', pp. 131–140. Copyright (2015) with permission of George Warman Publications (UK) Ltd.

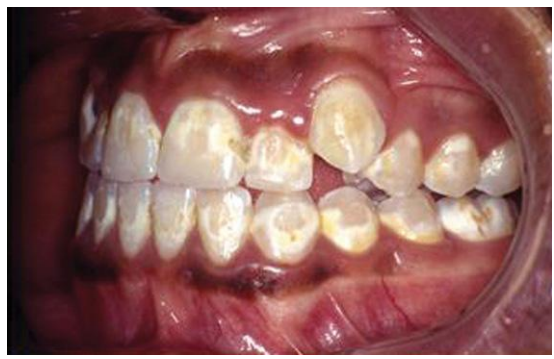


Figure 7.16 The devastating appearance of white spot lesion some weeks after removing a fixed orthodontic appliance in another patient with poor oral hygiene and a high sugar diet. The white spot lesions are now arrested, but they will remain as scars in the tissue.

- Avoid substances that make the dry mouth worse, including cigarettes and drinks containing caffeine and alcohol.
- Avoid astringent products because they will sting the mucosa. Thus, avoid strongly mint- or essential oil-flavoured mouthwashes, alcohol-containing products and strongly flavoured toothpastes. The patient will be aware of this

astringency and may ask your advice as to which products are bland. Toothpaste that does not contain sodium lauryl sulphate should be chosen because this is less harmful to the dry mucosa.

- Coat the lips with a lip salve or Vaseline.
- Chewing sugar-free gum will stimulate saliva if there is sufficient glandular tissue to stimulate. This may be helpful where medication is the cause of the dryness, but may not help if radiotherapy or Sjögren's syndrome is the problem because the glands are damaged.
- Oral hygiene should be immaculate—the teeth squeaky clean. A high fluoride-containing paste is sensible. These patients should be on regular recall with the hygienist, every 2 weeks until good cleaning is established and then every 3 months.
- The hygienist should apply fluoride varnish at these 3-monthly recall appointments.
- Try to help the patient restrict sugar attacks to mealtimes (three times per day). It is very difficult to avoid some sugar in a main meal, but between meal snacks should be sugar-free.
- Check any medication does not contain sugar and check antifungal agents as these have to be sucked.

A number of saliva substitutes are now available to make the patient more comfortable and to supply calcium, phosphate, and fluoride ions to counteract demineralization. Saliva substitutes have been produced in the form of sprays, lozenges, or mouthwashes. The anti-caries action of these products is largely unknown and clinical trials comparing their efficacy are lacking. However, their main benefit is to make the mouth feel more comfortable and the patient is the judge of this. It seems many patients prefer to take frequent sips of water, rather than use saliva substitutes. Only by trying products available in the particular country can the patient decide what works for them. The professional should check the product is not acidic because acid products will enhance dental erosion. 'Further reading' gives the reference for a UK Medicines Information document that can be consulted online to find acidic preparations best avoided in dentate patients.

Radiotherapy in the region of the salivary glands for a malignancy can produce an immediate mucositis and a long-term dry mouth because the salivary glands are damaged. Mucositis makes the mouth very uncomfortable during radiotherapy. A chlorhexidine mouthwash (10 ml of a 0.2% solution) can be useful if the mouth is too sore to clean with a toothbrush, and the mouthwash should be diluted with water if it is astringent. Add water to the 10-ml dose and rinse until the whole volume has been used.

Chlorhexidine is a bisbiguanide with antibacterial effects. There is a lack of evidence to support its use in caries prevention and it has local adverse effects:

- Discolouration of teeth, tongue, fillings, and dentures.
- Soreness and desquamation of the mucosa.
- Taste disturbances.

Thus, it would not be used for caries control, but can be very helpful on a temporary basis when the mouth is very sore.

7.6.5 Functionally-dependant adults

What does this term mean? Some individuals are disabled by illness, which may be physical (e.g. cancer and its treatment, stroke) or neurological (e.g. dementias, Parkinson's disease, multiple sclerosis). While many of these conditions are associated with old age, this is not always the case. These people may not be able to do basic things, such as wash themselves and clean their mouths. They are 'functionally dependant' on others to help them. They may be living at home with assistance from family or carers. Alternatively, in some cultures, for instance, in England and Denmark, they may live in residential or nursing homes.

We are ashamed to say that the oral health of elderly occupants of residential homes is very poor and surveys from Europe and USA have shown poor oral hygiene, high gingival indices, high denture debris levels, denture stomatitis, angular cheilitis, and a very high prevalence of coronal and root caries. This is not surprising because probably all of them are on multiple medications, which depress salivary flow so they have dry mouths. The medication may be in the form of syrup containing sugar, or a tablet may be crushed and mixed with jam to make it palatable. Carers should brush the teeth, but this is often not done, and some have said that they would rather wash bottoms than clean mouths. This situation is very sad indeed. The mouth is such a personal and intimate part of the body, it eats, it speaks, it kisses, it matters. In addition, the poor oral status may contribute to eating problems and poor nutrition. More importantly, the mouth may be the source of the organisms that can cause an aspiration pneumonia and premature death.

What can be done? Education programmes to teach carers to clean effectively have not been successful—their knowledge improves, but this is not transferred to changed behaviour. In addition, carers often change jobs and then the expertise is lost. Moreover, the economy is such that there are very few places, if any that can afford this. We are not used to accepting that elderly people require special care and personal support. Specific oral carers seem a reasonable suggestion, but to our knowledge this has not been tried and investigated in an experimental setting.

Each resident will have a care plan, which will include their nursing care and medications. We suggest an **oral care plan** should also be also obligatory and should include details of exactly how the mouth of each client should be handled. Toothpaste containing 5000 ppm fluoride may be used or a dental care worker should apply fluoride varnish every 3 months. Gloves and masks should be available for carers to use when brushing teeth. Electric toothbrushes would seem sensible if the client finds this acceptable. An opportunity to contact a member of a dental team who will attend when the carer perceives a difficulty is essential. Sometimes carers say the client will not allow their mouth to be touched. When this happens a dentist should check there is not an obvious source of pain in the mouth.

7.7 Guidelines

Public Health England has produced a guideline document entitled 'Delivering better oral health: an evidence-based toolkit for prevention'. Similarly, the Scottish Intercollegiate Guidelines Network has produced a national clinical guideline entitled 'Dental interventions to prevent caries in children'. Both these documents are available online and are referenced at the end of the chapter. They give the strength of evidence behind their recommendations and these brief documents will be of assistance.

While our advice concurs with their recommendations in almost all respects, there is one area where we would beg to disagree and would like to highlight this for consideration. It concerns the **use of fluoride varnish**. Both guideline documents suggest twice yearly application for **all children** aged 3 years to young adults. This **whole-population approach** to fluoride varnish is extremely costly, even when ancillary personnel are used, and while the authors totally concur that this is essential with regard to the use of fluoride toothpaste, they reserve the use of varnish for those who need it. Dental caries is not a result of fluoride deficiency. Fluoride toothpaste is available and should be used as described in this book, as an integrated part of proper oral hygiene combined with sugar restriction. It is a simple and rather cheap way of controlling dental caries. Remember, there is no clear evidence that any modality is more effective than any other—**make it simple and cost-effective.** This pre-supposes the dentist will spend the time to examine the teeth very thoroughly twice a year. **In populations where dental caries is well controlled, there is no need for biannual dental visits** (see Section 7.7.1, 'Recall intervals'). Children attending school dental services (see description of Danish dental services for children in Chapter 8) may be seen at regular intervals by dental assistants or hygienists and if they see any signs of lesion development they should be able to institute appropriate caries control). In adults, the English guidelines suggest varnish

should be applied for all giving concern (e.g. obvious active caries, orthodontic appliances, dry mouth).

The Scottish Government has also produced a guide for those who give oral care to older people. It is called 'Caring for Smiles' and is available online from www.healthscotland.com. For anyone involved in looking after this group of patients, this document will be invaluable. It is simply written, fully referenced, and contains forms for oral health assessment and oral care plans.

7.7.1 Recall intervals

We recommend a **highly individual recall interval**. There is no evidence to suggest a regular time interval for seeing patients again. If patients understand the message about caries control they will appreciate that they themselves are responsible for new caries lesion development—and progression of lesions. It is sensible, therefore, to ask the patient when they think they should be seen again. The dentist can and should recommend when the patient should be re-examined, **based on an individual assessment of the patient and their views.**

Most **adults with no active lesions** may be recalled at 1.5–2-year intervals. However, if there are **multiple active lesions** a shorter interval is needed, for instance 3 months. This can be to see a dental nurse/hygienist to check how the various non-operative treatments are going. In other words, to support the patient in their efforts.

If patients experience a **life crisis**, such as divorce, unemployment, loss of a family member, personal illness, the offer of seeing a dental nurse/hygienist at monthly intervals may be appropriate. **Elderly people** may likewise need more regular visits or home visits by oral health personnel to maintain the dentition. **Children and their parents** should also be offered individual recall intervals based on the caries status of the mouth. A child with early childhood caries should be seen again in 3 months after the initial course of treatment, whereas a child with no lesions would not need to attend again for a year. The exception here is the child with molars erupting. Regular visits to ensure the parent is able to clean the occlusal surface are very important.

Further reading

Carson, S. and Freeman, R. (2015). Fact or fiction: does a relationship exist between free sugars, dental caries and body weight? *Dental Update*, **41**, 862–866.

Fejerskov, O., Cury, J. A., Tenuta, L. M., and Marinho, V. C. (2015). Fluorides in caries control. In:Fejerskov O., Nyvad, B., and Kidd, E. A. M. (eds) *Dental Caries*, pp. 245–276. Wiley, Blackwell, Oxford.

Henderson, S. (2015). Saliva substitutes: choosing and prescribing the right product. UK Medicines Information (UKMi). 2013. Available at: http://www.evidence.nhs.uk/Search

?q=Medicines+Q%26A++190.8+Saliva+Substitutes%3A++Choosing+and+prescribing+the+right+product (accessed 9 December 2015).

Jawad, H., Hodson, N. A., and Nixon, P. J. (2015). A review of dental treatment of head and neck cancer patients, before, during and after radiotherapy: parts 1 and 2. *Br. Dental J*., **218**, 65–68, 69–74.

MacEntee, M. I., Bryant, S. R., Keller, H., Nguyen, C., and Yao, C. S. (2015). Caries control for frail elders. In:Fejerskov O., Nyvad, B., and Kidd, E. A. M. (eds) *Dental Caries*, pp. 321–322. Wiley, Blackwell, Oxford.

Moynihan, P. J. and Kelly, S. A. M. (2014). Effect on caries of restricting sugars intake - systematic review to inform WHJO Guidelines. *J. Dent, Res*., **93**, 8–18.

Nyvad, B. and Fejerskov, O. (2015). The caries control concept. In:Fejerskov O., Nyvad, B., and Kidd, E. A. M. (eds) *Dental Caries*, pp. 235–243. Wiley, Blackwell, Oxford.

Nyvad, B. and Kidd, E. A. M. (2015). The principles of caries control for the individual patient. In:Fejerskov O., Nyvad, B., and Kidd, E. A. M. (eds) *Dental Caries*, pp. 303–320. Wiley, Blackwell, Oxford.

Rogers, H., Morgan, A. G., Batley, H., and Deery, C. (2015). Why, what and how: caries control for erupting molars. *Dental Update*, **42**, 154–156. 159.

Scully C. (2014). *Scully's Medical Problems in Dentistry*, 7th edn. Churchill Livingstone, Edinburgh.

Guidelines

Public Health, England. (2014). Delivering better oral health: an evidence-based toolkit for prevention. Public Health England, London. Available at: https://www.gov.uk/government/publications/delivering-better-oral-health-an-evidence-based-toolkit-for-prevention (accessed 7 December 2015).

Scottish Intercollegiate Guidelines Network 138 (2014). Dental interventions to prevent dental caries in children. SIGN, London. Available at: http://www.sign.ac.uk/guidelines/fulltext/138/ (accessed 7 December 2015).

Caring for Smiles: Guide for Trainers. Available at: http://www.healthscotland.com/documents/4235.aspx (accessed 9 December 2015).

Chapter 8

Caries control in populations

8.1 Caries epidemiology

Up to this point, the scientific basis for caries control and practical details for delivery of caries control to the individual have been given. We now change tack and consider caries control in populations. In order to follow the health profiles in populations there is an important tool called **epidemiology.** This literally means 'the study of what is upon people'. It is derived from Greek where 'epi' means upon or among and 'demos' is people (population). In other words, epidemiology is the study of the distribution (how often) various diseases occur and why they appear in well-defined populations. It deals with groups of people, not individuals. Data thus obtained are used in public health for developing and monitoring strategies for health care in populations. Moreover, it can tell how diseases are influenced by hereditary factors, by physical and social environments, and human behaviour. All this helps health authorities to develop appropriate preventive interventions and make these as cost-effective as possible. In this chapter, having introduced the concepts of epidemiology, examples of caries control in two populations and its assessment using epidemiological measurements is given. However, the use of epidemiology has already been

described in Chapter 4, where Dean's observations on the relationship between fluoride in water supplies, the resulting dental fluorosis, and the concomitant caries reduction are described (see Chapter 4).

In a recording system of any disease it is important to have clear criteria for diagnosis. The following are important:

- How **valid** are the criteria of measurement? Do they record what they are intended to measure?
- How **reliable** are the criteria? Reliability is also covered by the terms **reproducibility**, and **consistency**. These terms imply that the same or different examiners can use the criteria in the same way on different occasions and obtain the same result.
- The criteria should be **clear**, **simple**, and **objective**. In other words robust. This is particularly important if manifestations of a disease are to be grouped in different categories of severity, as with dental caries.
- The criteria should be **quantifiable,** in numerical terms so that the disease manifestations in a group can be summarized and expressed by a distribution, a mean or a median, to allow for comparisons between groups.
- Criteria should be sufficiently **sensitive** to allow for a detection of shifts occurring in a population over time.
- Criteria should be **easy to learn** and fairly quick to use without unnecessary inconvenience for the individuals to be examined.

It is important to realize that there is no global consensus on the criteria for detection of dental caries. It has been known for a century that there are a full range of manifestations of dental caries lesions ranging from non-cavitated enamel lesions to lesions extending into the pulp. Despite this, there is a long-lasting tradition in caries epidemiology for recording lesions only when there is obvious cavitation. Data from the World Health Organization (WHO data bank) do not report on the pre-cavitated stages of dental caries and, therefore, these data give a very erroneous impression of the magnitude of the caries burden in many countries in most of the world. It is also unfortunate that often only data from children are reported. In most countries it is unusual to have representative data from adults and the elderly. An example of this problem can be seen in Figure 8.1 from a Kenyan population.

The arguments for not recording stages that reflect onset and pre-cavitation stages of caries lesions have been that these stages cannot be reliably diagnosed. This claim is not only erroneous, it is highly unfortunate because dentists and health workers are prevented in understanding how caries develops and progresses. For many years, this has prevented implementation of the caries control concept until the patients have cavities that will require drilling and filling as

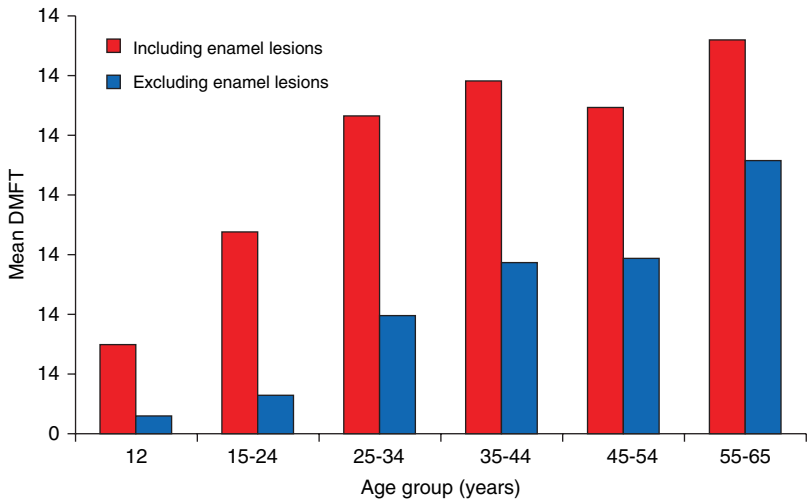


Figure 8.1 Mean DMFT scores in six different age groups in a Kenyan population in the mid-1980s. The data are presented with non-cavitated enamel lesions included and excluded. It is apparent that two entirely different views on the magnitude of the caries problems derive from such data. If one ignores enamel caries lesions and only record cavities (the WHO recommendation) the need for caries control in such populations will not be appreciated.

part of prevention in the classical context (see Chapter 4). In other words, this approach of ignoring the uncavitated lesion is an outdated concept linked to a false idea of what constitutes 'treatment' of caries. A very recent review of the global burden of untreated dental caries in both the permanent and deciduous teeth shows that in the last 20 years this great burden has remained fairly unchanged.

8.1.1 An index of measurement

The index of measurement of caries is the DMFT/S index where:

- D = decayed teeth.
- M = missing due to caries.
- F = filled.

The index may be used with teeth as the unit and, thus, in a fully dentate adult, data can be derived from 32 teeth (DMFT). It may also be assessed by **surface** (DMFS) and if each surface counts, adults will present five surfaces on molars and premolars (occlusal, mesial, distal, buccal, and lingual) and four surfaces

on incisors and canines (mesial, distal, buccal, lingual). This makes a total of 148 surfaces.

In the primary teeth lower case letters are used and the index is the deft/s where:

- d = Decayed teeth.
- e = Extracted due to caries and not just missing due to developmental reasons.
- f = Filled.

In the primary dentition data can be derived from 20 teeth or 88 surfaces if defs is to be used.

To derive a mean caries severity score for a group, the scores are summed and divided by the number of individuals.

The D/d component can be subdivided into categories depending on estimated depth of the lesions. Thus, D1 indicates an enamel lesion, D2 a lesion with a cavity in enamel, D3 a lesion with a cavity into dentine, and D4 a lesion judged to be into pulp.

It will be apparent that this DMFT/S index, although still the most commonly used, is associated with considerable flaws and may be difficult to use if comparing different populations. Here, are some of the problems:

- A score of 12 DMFT may reflect an individual with 12 open cavities, or with 12 fillings and no untreated lesions, or a person with say four open cavities, two missing teeth, and six filled teeth.
- In a low economy country, the D component will dominate, whereas in high economy countries, with a low ratio of dentists/population, fillings will be the main component. Now the index may tell little about need for operative treatment, but may rather reflect the number of dentists present and their restorative philosophy.
- The presence of restorations in surfaces may reflect when those dentists chose to place restorations.
- In addition, an occlusal extension of an amalgam restoration may have been placed to retain the filling, not because the surface was carious.
- Sealants are not recorded, and composite and resin restorations may be difficult to detect.
- Extractions may be influenced by dentists' view on treatment options and patient economy. This really matters because a missing tooth will count for four or five surfaces, and this could lead to an apparent overestimation of caries.

As long as the distribution of lesions in a population shows an even distribution, the mean DMFT/S can be a useful indicator of severity. Unfortunately, there are now more skewed distributions in modern society—a smaller number of individuals accounting for the majority of disease experience in a given

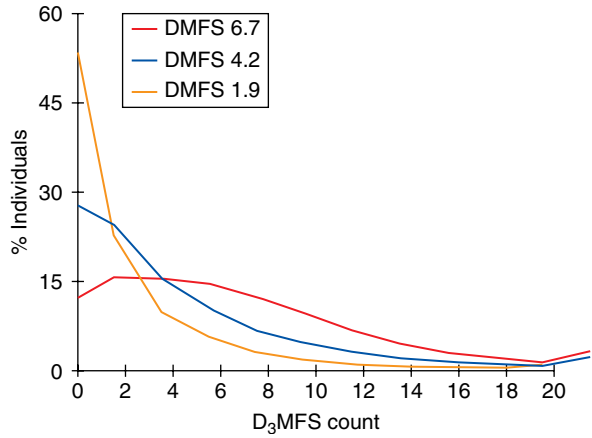


Figure 8.2 The frequency distribution of 15-year-old Danes, according to their individual DMFS scores recorded at cavity level. When the mean DMFS decreases from 1988 (red line), to 1993 (purple line), to 2012 (orange line), the distribution becomes very skewed. To explain this further, look at the red line, 1998 data. The line is relatively flat, the varying DMFS counts occurring in a similar number of individuals. Now look at the orange line, 2012 data. This is described as 'skewed' because most of the individuals have low DMFS and only about 10% have a DMFS over 3.

Reproduced from Baelum, V., and Fejerskov, O. (2015). How big is the problem? Epidemiological features of dental caries. In *Dental Caries: the disease and its clinical management* 3rd edn, Fejerskov O., Nyvad, B., and Kidd E., copyright (2015) with permission from John Wiley and Sons.

age group. Now the arithmetic 'mean' is meaningless! Compare Figure 8.2 and Figure 8.7 later in this chapter.

Chapter 3 discussed other recording systems such as Nyvad criteria and the ICDAS system. We recommended that the way of recording dental caries should have an immediate consequence for the way the patient is treated. There is little point spending time and money on surveys of the population unless the record is of benefit leading to better health and maintenance of a functioning dentition for life (see Epilogue).

8.2 Analysing caries epidemiological information

8.2.1 Definitions

To make sense of epidemiological studies, it is necessary to know the meaning of a number of terms.

Prevalence

Prevalence is the number of individuals in a given age group who at a specified time have experienced one or more caries lesions given as percentage.

This is different from **incidence**, which is the number of new caries lesions developed in a given time period in the specified population. The **incidence rate** is the rate at which new caries lesions occur where the numerator is the number of new lesions in a given period of time divided by the total number of surfaces at risk in the study population, expressed as a percentage.

Extent or severity

You may also be interested in the **extent or severity** of caries in a given population at a specified time. This reflects the mean number of teeth affected by caries of a given severity. We mentioned that D can be subdivided in D1 (non-cavitated), D2 (a cavity formed), D3 (a deep cavity without pulp involvement), D4 (with pulp exposure). Using this approach D2MFT would thus signify how many teeth on average in this population in question would have teeth with a cavitation, whereas D1MFT would signify all teeth with dental caries, irrespective of the severity in the population.

Sampling

Sampling selects a subsample of a population. It may be a **random** or non-random sample, and may be representative or non-representative. A random sample means that all individuals may have an equal chance of being selected—or if **stratified sampling** is used, the rate at which individuals from several subsets are sampled can be varied, so as to produce a greater representation of some classes than others.

Cross-sectional versus longitudinal studies

When the caries situation in a group is examined at a given period, cross-sectional caries data are collected. If a similar age group is then examined a decade later, it is possible to compare the different cross-sectional data sets and speculate if a difference is due to changing environmental factors (for example, dietary habits, fluoride exposure, more soft drinks and snacks available, more dentists). However, in a few studies, where it is possible to follow the **same** group of people over a decade, a true longitudinal study has been conducted. This can precisely inform what happens to dental caries as people grow older.

A randomized controlled trial

A randomized controlled trial (RCT) is an epidemiological experiment in which subjects in a population are randomly (by chance) allocated into groups, usually called 'test or study' and 'control' group(s) to receive, or not receive, an experimental intervention. This could be the testing of a toothpaste with a different concentration of a pharmaceutical agent where the 'control placebo paste' would not contain the agent. A **'blind'** trial implies that neither the patients

nor the dentists (called 'double blind') recording the outcome will know which paste is the control and which the test compound. It is evident that a double blind design is mandatory to avoid the examiner(s) from being biased. **Bias** implies that the experimenter(s) have an expectation, or prejudice, in favour of a certain outcome and, hence, the recordings of lesions may (non-intentionally) be influenced by this bias.

8.2.2 Some examples of study design

Consider some examples of the above definitions. For instance, how has the population in a given study been sampled? Is it representative of the whole population or does it reflect a selected group? Perhaps a given study deals with reasons for tooth loss in a country and then only reports on a sample of outpatients seeking pain relief at a dental hospital. The observations cover a highly selective group who have probably had toothache and not seen a private dentist for emergency care. This sample does not represent the whole population. It cannot be used to report the reasons for tooth loss in a whole country.

To state the obvious, children are not all alike. If researchers seek knowledge on the caries situation amongst children in a larger city, they should not just examine all children of a certain age group in a few selected schools. There will be large variations between groups of schools depending on which socio-demographic areas of town they come from. The sample must reflect children across the entire city. The extreme importance of socio-demographic areas will become apparent later in this chapter.

In any epidemiological study the age group(s) of interest must be defined. This is particularly important in caries epidemiological studies in children. Suppose children aged 9–12 years of age were to be selected for study. In this particular age range it is not possible to know how many permanent teeth have erupted. Some will have their second molars erupted and some will not. Thus, it is not possible to know the time the teeth been exposed in the oral cavity. For this reason, samples should not be taken according to school classes, but according to the age of the children. In any study, it is important to know the average number of teeth present, in other words, how many have been at risk. At the other end of the age spectrum, consider a sample of elderly people. Some will be edentulous so if reporting on caries, only dentate individuals should be selected.

Mean DMFT/S data will not be as informative as distribution data in this population. To give an example, consider studies on root surface caries prevalence. These studies are often difficult because usually this type of caries appears in a rather skewed distribution in a population. Is there, for example, any

association between the number of root surface caries lesions and the number of teeth present in the population? One might expect that the more teeth an individual has, the more likely it is that root surface caries will occur. However, studies have shown that if 60- or 70-year-olds are studied, there is only a positive association (more teeth, more caries) when patients have 1–9 teeth left! Those having more than nine teeth at this age, exhibit a negative correlation (more teeth left at this age means less caries). This means that the more teeth that have been maintained, the less likely they are to be at caries risk at later age. Remember from Chapters 1 and 2 that teeth are lost because of dental caries and restorative care predominantly.

8.2.3 Universal patterns of caries presentation

The relative susceptibility of teeth within the mouth follows a hierarchical pattern. In the permanent dentition, the first and second molars are the most caries susceptible, followed by the premolars. Lower anterior teeth are the least susceptible. The most caries prone surfaces are the pits and fissures followed by the approximal surfaces.

Despite the many criticisms of mean def/DMF data, these still give useful information in a population. Caries data at the population level follow certain universal patterns of occurrence:

- There is a mathematical relationship between the defs/DMFS and the mean deft/DMFT. For epidemiological purposes, it may not be necessary to record caries at surface level because sufficient information can be derived the deft/DMFT.
- When the mean caries experience decreases in a population, the percentage of 'caries-free' individuals will increase. The caries distribution within the population will, as a result, become more and more skewed (Figure 8.2). Note that the major differences are seen at the lower end of the range of DMF counts, but also the fraction of individuals at the high end 'the tail of the distribution' becomes smaller.
- When reading the term 'caries-free' please check to see the level of the diagnosis. If the pre-cavitated lesion was excluded, 'caries free' only means 'cavity free'. This could not be more important. It would be totally wrong to suggest people designated as having no cavities, need no treatment. If they have active non-cavitated lesions, they need to be exposed to the full range of caries control measures.
- There is a relationship between the caries level in the present population and the population caries level at later age. This means that the caries level at one age in a cohort of children will predict the caries level of that cohort at a later age. This applies both to groups and also at the individual level.

- When the mean caries experience recorded as deft/DMFT declines in a population the progression rate of dental caries through enamel decreases. Thus, with today's very low caries experience in many high-economy countries, there is no hurry for the dentist to start drilling and filling, but rather focus on caries control and arrest of lesions under development.

8.2.4 The relevance of age and gender

It is now well established that the caries experience in a population increases with age (Figure 8.3). This demonstrates that if no attempts are made to actively intervene with caries control, new caries lesions will continue to develop across all ages. For many years, however, caries was considered to be predominantly a problem in children and adolescents. This unfortunate misunderstanding arose from dealing with high caries prevalence populations and the use of the DMF/def indices. Most of the caries susceptible surfaces were already affected by the time the children were adolescents. Now the DMFT/S counts could not capture new caries development because most surfaces were either filled in countries with many dentists, or left untreated in populations without access to dentists. Moreover, few countries had the necessary data from adults.

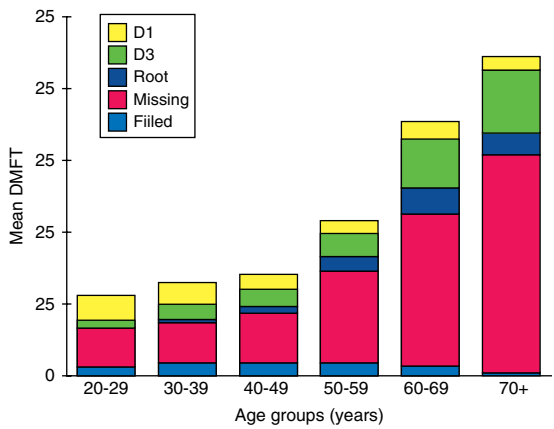


Figure 8.3 The mean DMFT and its components in six age groups of adult and elderly Chinese examined in 1985–86. This is cross-sectional data, but they clearly indicate how caries development and progression continues lifelong gradually resulting in tooth loss.

Reproduced from *Dental caries: the disease and its clinical management*, 3rd edn, Fejerskov, O., Nyvad, B., and Kidd, E., copyright (2015) with permission from John Wiley and Sons. Data from Journal of Dental Research, 68, 1989, Luan WM et al., 'Dental caries in adult and elderly Chinese', pp. 1771–1776.

In the 1980s different age cohorts were examined in both cross-sectional and longitudinal study designs in East Africa and China. It became obvious that not only do older groups have more caries experience than younger groups in the same population (Figure 8.3) but, more importantly, when the **same individuals** were followed for 10 years, it was possible to show that the caries incidence rates in the adult and elderly Chinese cohorts were the same. In other words, new caries lesions continue to develop with age, and this is irrespective of dealing with enamel surfaces or with the root surfaces, which gradually become exposed with age as the marginal gingiva retracts.

In general, females tend to have more caries experience than males. The explanation for this is not obvious, but perhaps they develop somewhat different dietary habits from a young age. In adults, the higher DMFT counts in women are a result of a higher number of filled or missing teeth, whereas men tend to have more untreated caries lesions. Women tend to visit dentists more often than men and this may explain these differences.

8.2.5 The relevance of race/ethnicity, social class, geography, and genes

The traditional definition of **race** refers to physical characteristics and has often been given a biological, inheritance interpretation. The concept of **ethnicity** covers broader cultural factors such as cultural traditions, a common ancestry, and origin. Ethnicity reflects groups which have distinctive features in their way of life, although they may also have a common genetic heritage making it difficult to separate the terms ethnicity and race. There is increasing recognition that 'race' is a social construct and that racial differences in dental caries experience may largely be attributable to income levels, educational background, employment status, and access to care in general. There is no evidence to support inheritance as a factor explaining the disparity in caries prevalence or incidence between different racial or ethnic groups.

On the other hand, there is a clear relationship between poverty status, and educational attainment and dental caries experience. This relationship applies to health in general, the poorer you are, the more likely it is that you will experience more bad health, diseases, and die younger than those in society who are wealthy and better educated. So the occurrence of dental caries in a population is related to measures of **socio-economic status**. In medium- and high-income countries, those with higher income, better education, better jobs, and living in attractive apartments or houses, have better oral health than those in society who are less fortunate. However, in low-income countries the reverse pattern may be seen with good examples being Vietnam and Kenya, where DMF counts increase with increasing income.

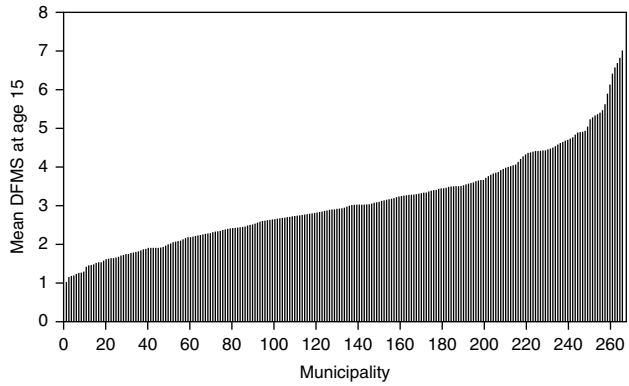


Figure 8.4 The within-country variation among 267 Danish municipalities in the mean DMFS (scored at cavity level only) in 15-year-olds in 2003. Note an eight-fold difference between the lowest and highest score.

Reproduced from *Dental caries: the disease and its clinical management*, 3rd edn, Fejerskov, O., Nyvad, B., and Kidd, E., copyright (2015) with permission from John Wiley and Sons. Courtesy of Dr Jens Heidmann.

The distinction between countries in terms of dental caries prevalence and incidence may, however, no longer be of so much interest because in today's world, it is possible to see extreme differences **within** many countries, particularly in the major cities. As people gather in huge cities there will inevitably be as great differences in oral health within cities as formerly, between poorer and richer countries. These within-city variations reflect the socio-economic disparity in populations. Thus, a mean caries score for a country may have little value when it is realized that there are as big differences within countries, as between countries, even in small countries like Denmark (Figure 8.4). A recent report from the Royal College of Surgeons shows big variations in different regions of England with respect to the dental health of 5-year-olds with about 20% of children in the South East, but 35% of children in the north-west having decay experience at the cavity level of diagnosis. These figures may indicate that different caries control strategies might be considered in the different regions of the country to cope with the oral health status in a cost–effective manner. May be much more innovative and profound suggestions are needed. We shall in the epilogue touch upon this because it requires a more elaborate explanation.

Familial patterns of caries occurrence have often been noted and given rise to claims of a **genetic** background. Patients often claim 'that my mother or father had also bad teeth'. However, when such situations are investigated, it seems that these families share a common environment. Families share dietary practices and oral hygiene behaviours. These two factors are sufficiently strong

determinants of the caries outcome to explain the notion of 'caries running in families'.

8.2.6 World Health Organization (WHO) database of caries in 12-year-olds

WHO has established a database that gathers information on oral health, including dental caries, where the age group is 12-year-olds. The information usually originates in locally initiated 'pathfinder surveys' carried out in accordance with a methodology described in the WHO manuals. These attempt some degree of standardization of methodology, such as simple diagnostic criteria, examiner calibration, and selection of study sites. This should allow for some comparability between studies. However, the sample sizes underpinning the estimates reported are often quite small and variation is considerable. The database must therefore be viewed as crude estimates associated with a probably sizeable degree of imprecision.

Despite these reservations, a pattern of dental caries occurrence among 12-year-olds emerges. The highest levels are generally seen in Eastern European countries varying from 2-5 D3MFT. Also Latin and South American countries have a fairly high level from 1.5 to 5 D3MFT. Western European countries are generally below 1 and so are most African countries except Gabon. In the Eastern hemisphere, China is also below 1 and Indonesia, Australia, New Zealand, Japan, and Korea range from 1 to 2 with the Philippines and India having from 3 to 4 D3MFT. Finally, the Middle East varies between 1 and 3, except Saudi Arabia, which hits the roof with a D3MFT of almost 6 by the age of 12 years.

8.3 Population studies of caries control

This chapter now presents some epidemiological caries data from a municipality in Denmark, where the dental care team has shown remarkable results by applying the caries control concept to a population. Throughout this book the biological rationale behind caries control has been provided and now it is time to show it in action with epidemiological data to validate results.

8.3.1 Dental health care for children in Denmark

A few words about how dental health care is organized for children in Denmark. Denmark passed a law on Municipal dental health care in 1972, whereby all children and adolescents up to the age of 18 years are offered free dental health care provided either by the public school dental health system or by private practitioners. At regular intervals, dentists have to provide the National Board of Health (NBH) with caries data on each child belonging to certain cohorts

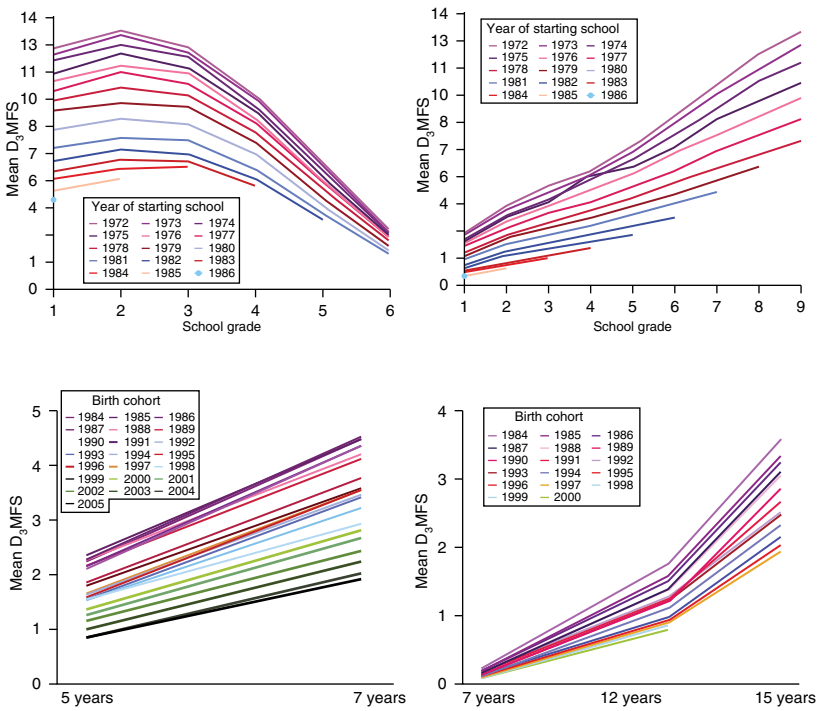


Figure 8.5 The unique data from the Danish Health and Medicines Authority regularly recording dental caries amongst all Danish children. The mean dmf/DMF counts are presented as cohort (a 'cohort' here describes any designated group of children who are followed over a period of time) and follow trend-lines, i.e. each colour reflects the same age cohort followed over time. The 2 upper figures show data (recorded at cavity level) from children starting school between 1972 and 1985 according to school grade. The primary dentition to the left and permanent dentition to the right. The 2 lower figures show to the left dmfs counts between the ages 5-7 year-olds for birth cohorts from 1984 to 2005. To the right mean DMFS counts are tracked from ages 7 to 15 year-olds for Danish birth cohorts from 1984 to 2000. The figures shows that caries increases with the age of the child. However, as the years go by, 1984–2000, the lines flatten out and children present in each new cohort with less dental caries. The caries status is improving steadily!

and the NBH can thus generate national dental health statistics of a unique nature shown in Figure 8.5. Since the 1970s the occurrence of dental caries has shown a steady decline and despite variations between the different municipalities as indicated previously, Denmark has experienced one of the world's largest documented reductions in dental caries (about a 90% reduction over the last

half of a century). One of the advantages of the unique health statistics is that individual municipalities can compare their DMF scores with the age-specific national mean values. The municipalities whose scores are higher than the national average values are likely to reconsider their strategies and priorities.

The explanation for the substantial improvement of oral health during this period is the overall improvement in socio-economic conditions in the population and the introduction of a large scale school dental health care with the establishment of dental clinics in many primary schools. We saw an extensive growth in number of school dentists paid by the public and an intensified focus on care for children, even in kindergarten (from 2 years of age). Up until the beginning of the 1980s these dentists organized fortnightly mouth rinsing programmes with 0.2% NaF solutions. As the occurrence of caries declined gradually during the 1970s, these rinsing programmes were gradually stopped because it became apparent that their efficiency (the cost–benefit ratio) was too low. Moreover, fluoridated toothpastes were recommended and by the beginning of the 1970s they dominated the toothpaste market. Professional application of a 2% NaF solution was also common on all tooth surfaces in the mouths of children who were considered to be at particular risk. This was done every 6 months, and later the solution was replaced by fluoride varnish treatments and fissure sealing.

It is important to emphasize that Denmark never introduced artificial water fluoridation and the natural waters in most areas contain between 0.1 and 0.4 ppm F–. Fluoride tablet regimes were introduced in the late 1970s in some municipalities, but as clinical studies demonstrated that they had no effect on dental caries, but only introduced dental fluorosis (this is exactly what happened in other studies from Holland), they never gained ground. The overriding concept has been not to build up complicated caries control programmes, but to keep them simple by focusing on oral hygiene and on the use of fluoridated toothpaste as their basic components. However, there is total freedom for the different municipalities to choose their own strategies for caries control and treatment.

For many years, it was not appreciated how important the filling component of the DMFT/S was when assessing the level of caries experience. In the early 1980s a study, on the radiographic diagnosis and clinical tissue changes in relation to the restoration of approximal carious lesions, showed that there was a restorative overtreatment in the school dental services. Non-cavitated lesions, often diagnosed only in enamel, or just into dentine on radiograph, were selected for filling. To argue for a more appropriate restoration threshold, the departments of Cariology and Restorative Dentistry of the two Danish dental

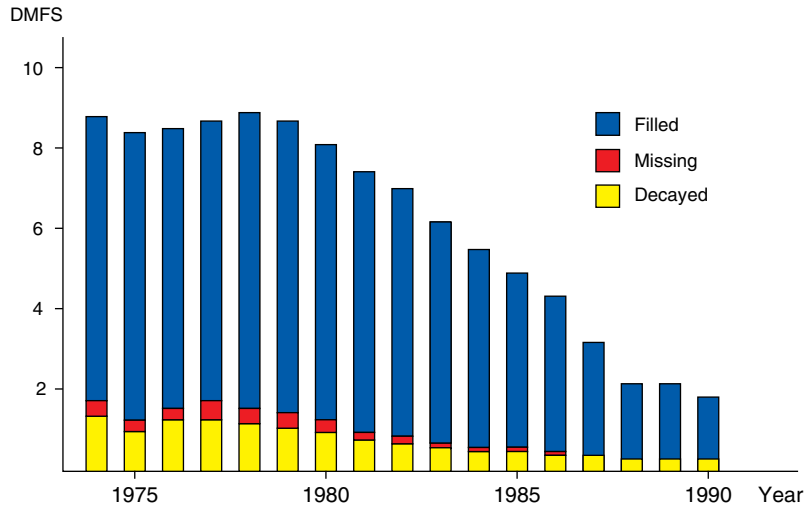


Figure 8.6 The mean DMFS in Danish 12-year-olds around the 1980s. Note how the dramatic decline in the 80s is predominantly the result of a change in the F component because of a change in criteria for restorative treatment decision.

Reprinted with permission from *Textbook of Clinical Cariology*, 2 edn, A. Thylstrup and O. Fejerskov, copyright (1994) with permission from Munksgaard.

schools joined forces to organize intensive courses for all dentists in the school dental services in an attempt to change their treatment criteria. The outcome of this was dramatic, a pronounced caries decline during the 1980s (Figure 8.6). During the 1990s and into the start of the twenty-first century, the caries levels continued gradually to decline. Of course, there have been periods of minor fluctuations, but in contrast with occasional claims from the international research community, there is no evidence of a return to a caries increase. The Danish population can therefore be considered as a low caries population.

However, it is still important to consider how dental caries may be further reduced in such populations and how the improved oral health can be maintained lifelong.

8.3.2 The Odder municipality dental health care programme

This section will describe the attempts made to introduce the caries control concept in a small municipality, Odder, south of Aarhus in Jutland over the last 6 years. In principle, the concept is based on the line of thinking advanced in the 2nd edition of the textbook *Dental Caries; the Disease and its Clinical Management*, and the general reflections presented in this book.

The municipality comprises around 21,500 inhabitants predominantly belonging to the middle class strata of the society. In the age group from 0 to 18 years of age there are a total of 5013 children and adolescents. In addition, to this age group the public service is also offered to disabled elderly persons (110 in total, predominantly living in care homes), as well as physically and/or mentally disabled persons (42). The service comprises diagnosis, caries control, and operative treatment. Moreover, up to 25% of children may receive orthodontic treatment.

To provide these services, the staff are a total of 3.3 dentists, 3.3 dental hygienists, and 10.1 dental assistants (fractions of a person represent part-time workers!). These individuals work together in teams so that two teams averaging 0.85 dentist, 1.2 hygienist, and 2.25 dental assistants are each responsible for about 2500 children. The dental teams were placed in two separate dental clinics. The entire school dental programme is coordinated by a dentist in charge of the services, assisted by two of the dental assistants.

In the early 2000s the caries experience was above the national average. A new dentist took charge in 2005 and analysed the dental records of some of the children having the most caries lesions. It became obvious that the predominant treatment had been filling, without any instruction in oral hygiene being recorded, apparently no dietary counselling, or recommendations on the use of fluoride-containing toothpaste.

The summary reports of two patients demonstrate this:

> *A girl born in 1989. Until the end of 2005 she had been to the clinic 90 times which included 40 examinations and operative treatment during 38 visits. Two primary molars had been filled 9 times before being extracted.*
>
> *A boy born in 1999. Until the end of 2005 he had visited the clinic 52 times. He had had 14 examinations and operative treatment during 30 visits. Two primary molars had been filled 8 times before being extracted.*

The analyses revealed that apparently little had been done to interfere with the ongoing disease processes, i.e. 'caries control', except excavating caries lesions and repairing previous restorations. Moreover, the focus on restorative procedures often resulted in dental anxiety amongst the children. The municipal dental service decided to apply the current theoretical knowledge about dental caries available in the above-mentioned textbook and formulated the following goals:

- In every age cohort **the percentage of caries-free children** should increase every year and the defs/DMFS scores should continue to decline to below the national values.

- At the **age of 18 years**, where children are leaving the public service, **they should predominantly have sound teeth or very few fillings**. This age group should by then have been trained in and have developed good oral hygiene habits. They should know about healthy dietary and drinking habits. They should have **no dental anxiety**.
- Parents should preferably participate in the visits to the clinics until the child has reached the age of 12 years.
- The communication with children and parents should focus on the **concepts of appreciative inquiry**. This means that the communication focuses on possibilities, rather than limitations and points out even the smallest positive changes.
- When leaving the public dental service, each individual should be carefully informed about his/her oral health status and the chosen private dentist is provided with records about past disease experience and caries control and operative treatment.

The role of each member of the dental team was specifically defined with these goals in mind so as to achieve the most cost-effective use of the resources:

- **The dentists** became team leaders and consultants. They were supposed to perform traditional restorative care **only when needed.**
- **The dental hygienists** became key persons as they were given the responsibility for most of the dental examinations, and they were taught to **'assess risk', i.e. to observe even the slightest signs of active caries lesions** and factors affecting their development, such as unhealthy oral health habits. Moreover, they were allowed to perform 'adjustments' of poorly accessible approximal areas between deciduous molars to allow for optimal oral hygiene performed by the child and the parents.
- **The dental assistants** were given an important role in caries control having their own patients, with responsibility for oral hygiene instruction, application of topical fluoride, sealing of surfaces if needed, and caring for children with anxiety for dental intervention.

Children along with their parents are invited **for the first meeting with the municipal dental service around the age of 1½–2 years of age.** A hygienist or dental assistant conducts an interview and instructs the parents, with a focus on the importance of oral hygiene (toothbrushing with fluoridated toothpaste and the use of dental floss), appropriate diet, and feeding habits.

All children are invited for examination at repeated intervals. At each examination special emphasis is put on:

- Assessment of oral hygiene (disclosing solution used).
- Signs of early caries lesions in enamel.

- Past experience on dental caries and its treatment.
- Dental caries experience amongst siblings.
- Eruption of teeth.

Based on this assessment an individual caries control plan is developed. This consists primarily of support for better oral hygiene (plaque control and recording plaque index), instruction in appropriate toothbrushing, always with a fluoride-containing toothpaste (twice a day) and use of dental floss (recommended use twice a week), topical fluoride treatment and, if needed, fissure sealing and diet counselling. **The intervals between these visits are highly individual** depending on the response of the child. **If the child is not considered to be at risk for developing caries lesions, the next recall is after 20 months.** Alternatively, if oral hygiene is very poor, the child and the parents might be seen again in a week or two to offer support and encouragement.

Concomitant with setting these specific oral health goals in 2006, the municipality established a working group in order to advance a general health policy. This group consisted of politicians, leaders of the elderly care and rehabilitation programmes, physicians, dentists, labour market representatives, and people from voluntary organizations. A health policy and an action plan were adopted by the City Council in 2007 and a general 'health coordinator' of the municipality was appointed. For the first 4 years the focus of the policy has been on diet and physical exercise. **A zero-sugar policy has been adopted for kindergartens and schools as part of the food policy by the municipality.**

The results of this basically simple general and specific dental health programme have been dramatic. From 2002 to 2012, the DMFS score amongst 15-year-olds dropped from around 3 to less than 1. In fact, this was the goal for 2015, which the dental service had already achieved by 2012. The percentage of caries-free children by the age of 15 was 67% in 2011 and 69% in 2012.

The target group, the 18-year-olds, shows a decline in DMFS from 6.6 prior to 2003 to about 1.5 by 2012. A 60–70% caries reduction had taken place in 8 years in a population already considered to belong to a 'low caries population'. Figure 8.7 demonstrates that 52% of the 18-year-olds left the public service with sound teeth in 2012, and only 5% had more than eight filled surfaces (the red category). It should be noted that this cohort has only been exposed to the new caries control concept for the last 6 years. Recent data from 2014 shows that the decline in caries continues steadily!

In addition to these quantitative results, questionnaires have shown that parents and children consider the municipal dental health care system as very supportive and positive, which adds to the impression, among the various members of the dental teams, of a mutual mission with success.

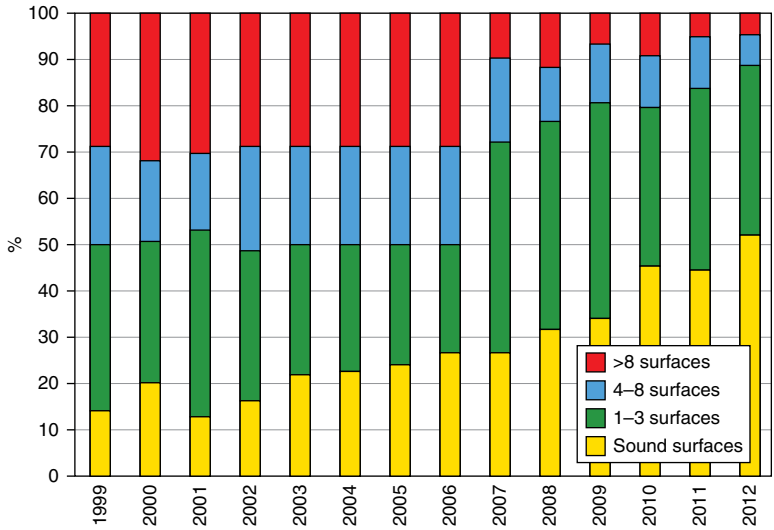


Figure 8.7 Data from Odder municipality dental service 1999–2012. The frequency distribution for all 18-year-olds in Odder municipality according to caries experience. The data show that the number of caries free individuals increase from 10 to 20% to above 50%, and the percentage of individuals with >8 surfaces treated diminish from about 30 to 5%.

Fejerskov, O., Escobar, G., Jossing, M., and Baelum, V. (2013). A functional natural dentition for all – and for life? The oral healthcare system needs revision. *J. Oral Rehabil.*, 40, 707–722.

You will notice that these impressive results were obtained by applying a simple caries control concept. There has been a total shift in the roles of the different members of the dental team, but no new recruitment of staff. One may argue that the population came from the middle class of society and certainly attempts should be made to analyse what constraints such efforts would meet when dealing with groups from the weakest socio-economic parts of society. The important message is, however, that in a population that was above the national average in caries experience, it has been documented that a 'significant' improvement in oral health can be achieved in a few years. No 'magic solutions', agents, or paint-on methods were needed. Hopefully, at this stage, it will be understood that such findings necessitate a profound rethinking of the role of classically-trained dentists in future societies if they truly wish to serve the dental needs of any population. Therefore, the book will finish with an Epilogue where some of the urgent questions that arise from these arguments are addressed.

8.3.3 A Scottish initiative: Childsmile

This chapter concludes with a brief description of Childsmile, a national programme funded by the Scottish government. The programme was developed in response to epidemiological studies that showed that in the 1990s, despite a caries decline in previous years, children in Scotland had some of the worst rates of dental decay in the UK. Inequalities in dental decay were also obvious with those from the lowest socio-economic groups bearing the greatest burden. For instance, social inequalities in health in the city of Glasgow have been among the widest in the world and the term 'Glasgow effect' has been used to express the high prevalence of some diseases in the city's populations. The Glasgow area contains almost half of the most deprived postcode sectors in Scotland.

In 1994, there was an obvious 'Glasgow effect' with 5-year-old children in Glasgow having more caries than 5-year-old children in the rest of Scotland. By 2012 there was no 'Glasgow effect'. In the Glasgow area the mean d3mft reduced from 4.3 in 1993 to 1.6 in 2012, and reduced from 3.0 to 1.3 in the rest of Scotland. The percentage of children with obvious decay reduced in Glasgow from 74% in 1993 to 36% in 2012 and reduced from 58 to 33% in the rest of Scotland. Importantly by 2012, Glasgow children and children in the rest of Scotland had similar caries levels when comparing 'like for like' by socio-economic status. This shows how important it is to cope with socio-economic inequalities in any attempt to promote better health in general. The disappearance of the 'Glasgow effect' corresponds with the implementation of the Childsmile programme, although whether this is, indeed, the cause, cannot be known for certain.

The Childsmile Core Toothbrushing programme was made available throughout Scotland during these years. This programme provides every child with a toothbrush, toothpaste, and oral health messages on at least six occasions by the age of 5 years. Children also receive a free-flow feeder cup by the age of 1 year. This cup allows the child to drink normally as opposed to sucking on a teat attached to a bottle.

Every 3- and 4-year-old child attending nursery, is eligible to be offered free, daily, supervised toothbrushing within their nursery establishment. Supervised toothbrushing is also offered to 6- and 7-year-old children in at least 20% of local authority primary schools situated in the most deprived areas. Children who attend nurseries, schools, childminders, and after-school clubs should be offered healthy snacks and drinks as part of national initiatives to improve dental health and reduce childhood obesity.

'Childsmile practice' is another facet of the programme where a 'health visitor' reinforces oral health messages and encourages registration with a dentist by 6 months of age. Alternatively, the family can be referred to a 'dental health

support worker' who contacts the family when their child is about 3 months old, advising on how to care for first teeth and find dental services. For the most vulnerable, a long period of home support may be needed before they will engage with dental services. 'Extended duty dental nurses' are part of Childsmile practice, and are trained in oral health promotion and fluoride varnish application (twice yearly from 2 years of age). Their role is to support the dental team in Childsmile care.

Two other aspects of Childsmile are the nursery (3–4 years) and school (from 5 years) programmes. These are also targeted at the most deprived areas and include twice- yearly fluoride applications.

Further reading

Blair, Y. I., McMahon, A. D., Gnich, W., Conway, D. I., and Macpherson, L. M. D. (2015). Elimination of 'the Glasgow effect' in levels of dental caries in Scotland's five-year-old children: 10 cross sectional surveys(1994–2012). *BMC Publ. Hlth*, **15**, 212.

Baelum, V. and Fejerskov, O. (2015). How big is the problem? Epidemiological features of dental caries. In:Fejerskov, O., Nyvad, B., and Kidd, E. (eds) *Dental Caries. The Disease and its Clinical Management*, pp. 21–41. Wiley Blackwell, Oxford.

Fejerskov, O., Escobar, G., Jossing, M., and Baelum, V. (2013). A functional natural dentition for all - and for life? The oral healthcare system needs revision. *J. Oral Rehabil*., **40**, 707–722.

Kassebaum, N. J., Bernabé, E., Dahiya, M., Bhandari, B., Murray, C. J. L., and Marcenes, W. (2015). Global burden of untreated caries. A systematic review and metaregression. *J Dent Res*, **94**, 650–658.

Macpherson, L. M. D., Anopa, Y., Conway, D. I., and McMahon, A. D. (2013). National supervised toothbrushing and dental decay in Scotland. *J. Dent. Res*., **92,** 109–113.

Epilogue

What are the consequences for dental manpower of providing effective oral health?

In the first chapter we addressed the question 'Why do we lose teeth?', and showed that dental caries and its consequence—dental restorations—represent the majority of the work that occupies most dentists, whether in private or public dentistry. Oral health is more than dental caries and its sequelae, but now we can demonstrate how and why dental caries can be controlled in all age groups by simple means. This will profoundly influence the dental workforce required in any society in the years ahead.

Our readers are a young generation—students of dentistry, hygiene, and other oral health care workers. You cannot read and appriciate the messages in this book without asking, 'How then do we best serve the population?'. Therefore, we decided, in this last section of the book, to raise some questions and hopefully identify some of the challenges that we believe are facing the profession. This is not because we are going to criticize the development in oral health over the past 50 years in various parts of the world. Quite the opposite; there has been an unprecedented decline in dental caries in some parts of the world that dentists have contributed to. However, it is apparent that increasing the number of dentists does not reduce oral diseases. This is documented in several countries in different parts of the world. More dentists result in more and more technically-advanced restorative treatments offered to those who can afford it, but leave the major parts of populations—often those having socio-economic challenges—without the help that can be provided.

We have to ask ourselves if we are truly trying to address the need of the populations or, in reality, the need of the profession. By training more dentists, is a need created that is not based on medical problems? For instance, the development of so-called 'cosmetic dentistry'—does this really justify the training of a 'classical' dentist? Consider fissure sealing, vanish applications, bleaching, polishing and scaling, just to mention what some dentists may spend time on. Do these procedures justify the claims that more dentists need to be trained?

In some countries, it is claimed that lack of money, manpower, and specialist-training is the problem in the battle against dental caries in children. Is this correct? We sincerely doubt it. The health sector in most countries is becoming extremely expensive. There is a change in the demographic profile in

populations all over the world with a rapidly growing number of elderly people in need of care and, often, advanced medical treatments. We would dare to say that dental services cannot expect to get a better share of the total health budget. So we advocate that, for the amount of money presently allocated to oral health care, it should be possible to provide a much more cost-effective oral health care system for the entire population, **if current knowledge on the aetiology and oral disease developments were to be applied** in cost-effective disease control.

We suggest this will profoundly alter the dental workforce required if implemented in society. Are more dentists trained in a traditional concept of restorative dentistry needed? The health personnel required to carry out the non-operative caries control measures are not dentists, but oral health educators, hygienists, and possibly specially-trained dental assistants, although there must be unease that, if these ancillaries get a drill in their hands, they may also be tempted to start the 'drilling and filling' scenario.

We totally accept that there is a generation of older people, with heavily restored mouths, who require complex restorative treatments, the so-called heavy metal generation, but in many countries there are already sufficient dentists to care for them. In countries such as Colombia there are now about 60,000 dentists (a ratio of 1 dentist/750 persons) and, despite this, there is no effect on the level of oral health! It is inconceivable that dentists, with their technically-advanced restorative training, are going to be happy to spend a practicing lifetime carrying out the non-operative treatments required for contemporary dental health. From a health-economic perspective, this group have the wrong training, gained at great expense to themselves and/or their countries. We have had it explained to us, politely but firmly in wealthy countries, that the non-operative approach just will not earn sufficient money to repay dental student debts, let alone earn a future living. Not only are dentists not cost-effective for oral disease control, they do not hold the correct competency profile to serve the needs of major sections of populations.

So let us put an alternative for consideration. Look at the lovely drawing by Whistler (see Figure 9.1) and turn it upside down. May be the same should be done with the way oral health care should be organized in the future. Oral health care providers (OHCPs) are needed with a different competency profile to today's dentist. This group would major in the non-operative treatments and behaviour management techniques, particularly for those groups at present underserved, such as the socially-deprived and functionally-dependant groups, both the very young and the very old. OHCPs should be at the centre of health service provision at local level. They should be responsible for managing dental

Figure 9.1 Rex Whistler drawing.

teams (dental assistants, oral health educators, hygienists, therapists) all working to meet the needs of most individuals, families, and communities. The dental teams should have most of their personnel comprised of these ancillary staff with responsibilities for disease control.

The OHCPs should plan health care strategies and set priorities at local level so that those in need get priority. They should be trained in simple dental restorative care and be able to refer patients in need of more complex advanced treatments to clinics in nearby hospitals or major comprehensive health clinics, where there are 'dentists' trained in oral rehabilitation, working as an integrated part of general health care together with other medical specialities. Thus, the OHCPs should be competent and skilled not only in the diagnosis and control of oral diseases, but most importantly in general public health, basic health economy, team management, and communication. The OHCPs and their staff should be able to cater for the oral health care needs of the great majority of the population, and be gatekeepers with respect to advanced oral health care needs. Their services must be integrated into the general health care

systems. A change such as the one suggested should be able to be carried out gradually without additional expenditure to society. This would require a thorough rethinking of the dental profession as we know it today, but we believe that the time is ripe!

Edwina Kidd and Ole Fejerskov
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